A STUDY ON EVALUATION AND OUTCOME OF IMPAIRMENT OF CONSCIOUSNESS AND COMA IN PEDIATRIC POPULATION

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CERTIFICATE

This is to certify that this dissertation entitled '**Evaluation and Outcome of Impairment of Consciousness and Coma in Pediatric Population**' submitted by **Dr.V.Ramakrishnan** appearing for D.M.,Degree examination in August 2011 is a bonafide record of work done by him under my direct guidance and supervision in partial fulfillment of regulations of the Tamil Nadu Dr. M.G.R. Medical University, Chennai -32. I forward this to the Tamil Nadu Dr.M.G.R.Medical University, Chennai.

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This is to certify that the INSTITUTIONAL ETHICAL COMMITTEE OF TIRUNELVELI MEDICAL COLLEGE AND HOSPITAL, TIRUNELVELI-11 has unanimously approved the dissertation titled '**Evaluation and Outcome of Impairment of Consciousness and Coma in Pediatric Population**' by **Dr. V. Ramakrishnan D.M** (Neurology) Post Graduate student of Tirunelveli Medical College, Tirunelveli is its meeting held on 09-10-2010.

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CONTENTS

INTRODUCTION

Coma is a relatively common condition in the paediatric intensive care unit. Epidemiological studies generally divide the causes of coma as traumatic and nontraumatic. The incidence of non traumatic coma was five times greater in children in developed countries. Non traumatic coma is an important source of morbidity and mortality in paediatric age group. Design of appropriate and efficient protocols of investigation for coma require an understanding of the relative frequencies of various potential etiologies. There are no prospective study on the etiology, clinical signs, severity of non traumatic coma in children with a view to define predictors of outcome in south Tamilnadu. Considering the fact that acute non traumatic coma is accounting for 10 to 15% of all hospital admissions, there is a need for more studies so that outcome rather than survival can be improved.

Consciousness is a state of normal cerebral function in which the child is aware of both self and surroundings and responds to stimuli, both internal, e.g., hunger and external. Sleep is a normal variation of this state. Impairment in consciousness may range from confusion, drowsiness and stupor to coma and brain death.

The ascending reticular activating system (RAS) is a collection of neurons located in the brainstem and medial thalamus, having connections with all parts of the cerebral hemispheres. This system maintains the brain in wakeful consciousness. Impairment of consciousness and coma occurs due to lesions that damage the RAS or its projections, disruption of large portions of both cerebral hemispheres, suppression of reticulo-cerebral functions by drugs, toxins, inflammation, or metabolic derangement etc.

Most cases of acute impairment of consciousness and coma are due to potentially treatable conditions and respond well to treatment. In certain conditions prognosis depends more on the causative illness rather than the severity of the illness. Outcome also depends on the speed and quality of the treatment given. Residual neurological deficit might improve over time with proper physiotherapy and rehabilitation. Cognitive functions may improve over time. Behavioral problems are common during recovery which may also improve in due course.

REVIEW OF LITERATURE

The study of consciousness represents one of the oldest areas of neuro-science. Since the days of the Greeks, men have known that normal conscious behavior depends on intact brain function and that disorders of consciousness are a sign of cerebral insufficiency. William James, in 1890, wrote that the cortex is the sole organ of consciousness in man (James, 1890). Nonetheless, reconciliation of the concept of mind (consciousness and awareness) and the structure and function of the brain has long been elusive (Sperry, 1965). Evidence that there is a neural correlate of consciousness, however, is clearly established {Crick and Koch, 1998). Multiple Philosophical, metaphysical and psychologic theories of consciousness have been elucidated (Zeman, 2001). An early step in neurobiologic understanding of consciousness and its alterations was the identification of brainstem structures essential for cortical activation [Damasio, 2003; Moruzzi and Magoun, 1949; Neylan, 1995]. Recent theories that the brain's electromagnetic field is the equivalent of the conscious mind allow reconciliation of the mindbrain problem and suggest methodology to scientifically study consciousness and alteration of consciousness at the neurophysiologic level [John, 2001, 2002; John et al., 2001; McFadden, 2002a, 2002b]. Because neurobiologic processes that are realized in brain structures are responsible for consciousness

[Baars et al., 2003; Neylan, 1995; Searle, 2000] neurologic disorders of the cortex and its brainstem activators result in impairment of consciousness and coma. Definitions of consciousness, impairment of consciousness, coma and related states have proposed throughout the history of medicine and have been reviewed, refined, and stated systematically during the past several decades[Ashwal, 196; Ashwal and Cranford, 2002; Bates 1993; Bozza Marrubmi, 1984; Medical Research Councial Brain Injuires Committee, 1941 Michelson and Ashwal, 2004; Plum and Posner, 1982]. Understanding neurological diseases depends on the clinical assessment and interpretation of consciousness, the content of consciousness, and alteration of consciousness. Evaluation of consciousness in the pediatric patient must take into account age and the appropriate developmental level[1,2]. The, diagnosis of coma and other impairments of consciousness involve both state and reactivity.

Along the continuum from normal consciousness to coma or unarousable unconsciousness, many terms are used to describe mental state and reactivity. When there is doubt about the appropriate use of one of these terms, it is far better to describe the state and reactivity rather than label it.

RETICULAR FORMATION [4]

The reticular formation consists of a collection of nuclei that forms the central core of gray matter throughout the brain stem. Although as many as 20 nuclei can be differentiated by their cytoarchitecture, connections, and functions, they belong to two fundamentally different longitudinal zones, medial and lateral.

THE LATERAL ZONE OF THE RETICULAR FORMATION IS RECEPTIVE AND INTEGRATIVE

The lateral zone of the reticular formation contains many interneurons with short axons. These interneurons integrate reflex connections between the sensory and motor cranial nerve nuclei and receive inputs from long tracts from the spinal cord and the forebrain. In this capacity, the lateral zone of the reticular formation can be viewed as the rostral extension of the interneuronal pool of the spinal cord.

THE MEDIAL ZONE OF THE RETICULAR FORMATION GIVES RISE TO ASCENDING AND DESCENDING TRACTS

The medial zone contains many large neurons with extensive axonal projections. Collectively, these cells receive information from the lateral zone interneurons and project to the spinal cord, cerebellum, hypothalamus, and cerebral cortex, especially the limbic lobe cortex.

Some very large neurons with bifurcating ascending and descending axons reach both the hypothalamus and the spinal cord. Other neurons with only ascending or descending axonal projections nonetheless inter-connect with each other within the reticular formation, so they also contribute to the distribution of its influence along the entire neuraxis.

Nuclei of the reticular formation function prominently in the processing of pain, visceral function, posture and muscle tone, and eye movements. Nuclei of the reticular formation also contribute to behavioral arousal and participate in controlling cycles of sleep and wakefulness.

ASCENDING REFICULAR ACTIVATING SYSTEM AND AROUSAL

The reticular formation participates with a larger system, the **ascending reticular activating system,** in processes required for alertness or **arousal.** Specific nuclei of the anatomically defined reticular formation, most of which use acetylcholine as a neurotransmitter, belong to the ascending reticular activating system. Through their projections to the glutamatergic thalamocortical neurons in the **intralaminar nuclei of the thalamus,** these reticular formation nuclei activate the cerebral cortex and initiate or increase arousal.

The cholinergic neurons of the reticular formation also project to other cholinergic nuclei in the forebrain, including the basal nucleus of Meynert, that, in turn, excite the cortex. Finally, monoaminergic neurons of the brain stem contribute to arousal by direct stimulation of the cerebral cortex. These include dopaminergic projections from the ventral tegmental area, noradrenergic projections from the locus ceruleus, and serotonergic fibers from the raphe nuclei. Thus, five different neurotransmitter systems (glutamatergic, cholinergic, dopaminergic, noradrenergic, and serotonergic) contribute to cerebral cortical excitation in behavioral arousal.

SEROTONERGIC AND NORADRENERGIC CELL GROUPS

Two neurochemically defined groups of nuclei within the central core of the brain stem function as part of the reticular formation. These include the **serotonergic nuclei,** located along the mid-line **raphe** of the brain stem, and the **noradrenergic celi groups,** which include the **locus ceruleus** and cell groups in the medial reticular zone of the pons and medulla. These two monoaminergic systems originate in the brain stem and in no other part of the central nervous system.

Similar to other medial reticular formation neurons, both serotonergic and noradrenergic neurons give rise to very long axons. In addition, both these neuronal groups distribute fibers to all parts of the

neuraxis, from the caudal spinal cord to the cerebral cortex. Nevertheless, selective projections arise within each group of nuclei. Within the serotonergic nuclei, for example, the most rostral cell groups send axons to the cerebrum, the most caudal send axons to the spinal cord, and the intermediate nuclei influence cranial nerve nuclei and the cerebellum . In the noradrenergic nuclei, the locus ceruleus sends axons to far rostral and far caudal central nervous system targets as well as the brain stem and cerebellum. The pontine and medullary noradrenergic cells have more limited projections, with notably less influence on the cerebral cortex.

The monoaminergic projections serve many different functions, which are as diverse as modulating sensory transmission, states of alert-ness, and mood. In all their target tissues, the monoamines play a modulatory role, by enhancing or decreasing the responsiveness of the neuron pools they innervate

SLEEP AND WAKING[4]

Sleep consists not simply of the absence of arousal. It is an actively induced behavioral state essential for life. In addition, sleep occurs in cycles that have different phases, each of which includes a particular pattern of physiologic states. In humans, two general phases of sleep, **slow-wave sleep** and **rapid-eye-movement (or REM) sleep,** differ dramatically in their characteristic levels of electroencephalographic

activity, skeletal muscle tone, parasympathetic tone, and ease of reversibility (ease with which the individual can be awakened). Further, both these phases of sleep differ from the physiologic pattern of waking state functions.

Numerous neuroanatomic sites regulate specific aspects of sleep and waking. These sites are located primarily in the hypothalamus, the mid-brain and pontine reticular formation, and the aminergic cell groups of the brain stem.

The activity of cholinergic and associated noncholinergic neurons in the midbrain reticular formation maintains the **waking state.** These neurons project to the thalamus, where they interfere with a slow-wave-sleep bursting pattern of firing in the thalamus and cortex. The slow-wave-sleep bursting pattern blocks the transmission of sensory information to the cortex. A group of histaminergic neurons in the posterior hypothalamus also becomes active during wakefulness.

The nucleus reticularis pontis oralis, which extends from the pons into the caudal midbrain, assumes particular importance in both **waking** and REM sleep states. Several functional groups of neurons within this nucleus interact with nearby histamine neurons in the posterior hypothalamus, serotonergic raphe neurons, and noradrenergic cells of the locus ceruleus. The interactions of these cell groups influence waking and

also control the visual system spiking activity, rapid eye movements, and loss of skeletal muscle tone that characterize REM sleep. Gamma-aminobutyric acidergic neurons in the region of the anterior hypothalamus induce slow-wave sleep by inhibiting histaminergic neurons of the posterior hypothalamus and cell groups in the . nucleus reticularis pontis oralis that sustain wakefulness.

DEFINITIONS

Consciousness is the spontaneously occurring state of awareness of self and environment. Consciousness has two dimensions - wakefulness and awareness. Awareness requires wakefulness but wakefulness can be present without awareness. **Clouding** of consciousness is minimal reduction of wakefulness or awareness wherein main difficulty is attention or vigilance[5,8]. **Confusion** is the state of impaired ability to think and reason clearly at developmentally and intellectually appropriate level. Other definitions of impairment of consciousness can be divided into the following categories:

- Impairment of consciousness with activated mental state
- Impairment of consciousness with reduced mental state
- Impairment of consciousness along the continuum of coma-vegetative state-minimally conscious state and related conditions[(5,8).]

IMPAIRMENT OF CONSCIOUSNESS WITH ACTIVATED MENTAL STATE

- Hallucinations are perceptions of sensory inputs that are not present; illusions are misinterpretation of actual sensory stimuli; delusions are incorrect thoughts that do not change when challenged by contradictory evidence or logical reason. Delirium is an activated mental state that may include disorientation, irritability, fearful response, and sensory misperception. Delirium will more likely involve both cerebral hemispheres than one side of the cerebrum or brainstem alone[13,19].

IMPAIRMENT OF CONSCIOUSNESS WITH REDUCED MENTAL STATE

- **Obtundation** is mild to moderate reduction in alertness with decreased interest in surroundings and slower than normal recovery to stimulation.

Stupor is a state of unresponsiveness little or no spontaneous movement resembling deep sleep from which the patient can only be aroused by vigorous and repeated stimulation. The patient goes to pre-stimulation state without continuous stimulation.

COMA

Coma is a state of deep, unarousable, sustained pathologic unconsciousness with the eyes closed that result from the dysfunction of the ascending reticular activating system in the brainstem or both cerebral hemispheres. Coma usually requires the period of at least 1 hour to distinguish coma from syncope, concussion, or other states of transient unconsciousness.

Table:1. Modified Glasgow coma scale[(25)].

	> 5 Years	< 5 years
Eye opening		
4 3 2 1	Spontaneous To voice To pain None	
Verbal 5 4 3 2 1	 Orientated Confused Inappropriate words Incomprehensible No response to pain	 Alert, babbles, coos, words or sentences – normal Less than usual ability, irritable cry Cries to pain Moans to pain No response to pain

Motor		
6	Obeys commands	Normal spontaneous movements
5	Localises to supraocular pain (>9months)	
4	Withdraws from nailbed pressure	
3	Flexion to supraocular pain	
2	Extension to supraocular pain	
1	No response to supraocular pain.	

Despite its limitations for not taking into account the important brainstem reflexes like pupillary reactivity or oculocephalic or oculovestibular reflexes, the modified GCS for children is useful for objective assessment of comatose pediatric patient.

The sum total score of GCS tells little as compared to the individual responses like ocular, verbal or motor. Short-term survival can predicted by ocular and motor responses. In addition, the absence of one or more brainstem reflexes have adverse short-term outcome.

In the long-term prediction of outcome in acute non-traumatic coma, MGCS is not useful. However, verbal response, a component of MGCS, correlates well with long-term functional outcome and intelligence quotient.

DIFFERENTIAL DIAGNOSIS OF COMA

Coma needs to be differentiated from other states of altered consciousness as the prognosis and management in each case will differ. Given below is a table (Table 2) to differentiate between coma and other states of altered consciousness

Table: 2 Comparison of clinical features associated with coma, vegetative state and locked-in syndrome

Condition	Consciousness	Sleep wake	Motor function	Auditory function	Visual function	Communication	Emotion
Coma	None	Absent	Reflex and postural responses only	None	None	None	None
Vegetative state	None	Present	Postures or withdraws to noxious stimuli	Startle	Startle	None	None
			Occasional nonpurposeful movement	Brief orienting to sound	Brief visual fixation		Reflexive crying or smiling
Minimally conscious state	Partial	Present	Localizes noxious stimuli	Localizes sound location	Sustained visual fixation	Contingent vocalization	Contingent smilling or crying
			Reaches for objects	Inconsistent command following	Sustained visual pursuit	Inconsistent but intelligible verbalization or gesture	
			Holds or touches objects in a manner that accommodates size and shape				
			Automatic movements (e.g., scratching)				
Locked-in syndrome	Full	Present	Quadriplegic	Preserved	Preserved	Aphonic / anarchic Vertical eye Movement and Blinking usually Intact	Preserved

CAUSES

The causes of coma in children can be divided into three distinct but overlapping categories[2,4].

- Infectious or inflammatory
- Structural
- Metabolic, nutritional or toxic

Table :3 Etiology of coma

Infectious or Inflammatory	Structural	Metabolic, nutritional toxic
A. Infectious Bacterial meningitis Viral encephalitis Rickettsial Infection Protozoan Infection Helminthic Infection	A. Traumatic Concussion Cerebral contusion Epidural hematoma Or effusion Intracerebral Hematoma Diffuse axonal injury Shaken baby Syndrome	A. Hypoxic Ischemic Encephalopathy Shock Cardiac or pulmonary failure Near drowning Carbon monoxide poisoning Cyanide poisoning Strangulation
B. Inflammatory Sepsis – associated Encephalopathy Vasculitis, Collagen Vascular disorders Demyelination Acute disseminated Encephalomyelitis Multiple sclerosis	B. Neoplasm	B. Metabolic disorders Sarcoidosis Hypoglycemia Fluid and electrolyte imbalance Endocrine disorders With acidosis Diabetic ketoacidosis

		Aminoacidemias With hyperammonemia Hepatic encephalopathy Urea cycle disorder Disorders of fatty acid metabolism Reye's syndrome Uremia Porphyria Mitochondrial disorder Leigh syndrome
	C. Vascular disease Cerebral infarction Thrombosis Embolism Venous sinus thrombosis Cerebral hemorrhage Subarachnoid hemorrhage Av malformation Aneurysm Tramua to carotid or Vertebral arteries in the neck	C. Nutritional Thiamine deficiency Pyriodxine deficiency Niacin or nicotinic acid deficiency pyridoxine dependency folate and vitamin B12 deficiency
	D. Focal infection Abscess Cerebritis	D. Exogenous toxins and poisons
	E. Hydrocephalus	E.Hypertensive encephalopathy
		F. Burn encephalopathy

(Source : Swaiman and Aishwal, Pediatric Neurology, Principles and Practice, 4th Ed; 1384).

EVALUATION

The evaluation of a comatose child focuses on urgent evaluation of neurological parameters which is followed by a more detailed neurological examination to identify the cause and location of neurological injury.

The clinical approach to impairment of consciousness and coma must be comprehensive and systematic. Few questions needs to be asked and answered while doing the quick neurological assessment[3,4,11].

1. Is the patient unconscious; if so, how deeply?

This is the most important question of all and may well be the most difficult to answer. The Glasgow coma scale was designed to assess depth of coma after head injury in adults and has been used in pediatric non-traumatic coma (refer above). At initial presentation, it is preferable to err on the side of recording too low a score, as it is easier to withdraw treatment from a child who is improving than to resuscitate one who deteriorates.

2. Is the intracranial pressure raised?

For an unconscious patient, the time to ask oneself whether or not is intracranial hypertension is as soon as this basic triage is done, as irreversible brain damage may supervene long before it is possible to measure the intracranial pressure (ICP). In almost all cases whether the etiology is infectious or otherwise the answer is yes.

The important steps are: (i) to memorize the stages of progressive herniation which are compatible with intact survival (in bold in tables 2 and 3); (ii) to acquire the habit of serially examining the patient's conscious level (Table 1) and brainstem reflexes (Table 3) with these concepts in mind, so that progression is recognized immediately; and (iii) to learn the management algorithm so that action is taken as swiftly as possible.

3. What is the emergency management of the unconscious child?

The potential life-saving maneuvers might be different from the long-term management of these children. The main priority in these children is to maintain airway breathing and circulation. A child might be in shock esp with meningitis. Timely correction of glucose level in a hypoglycemic child might be lifesaving. A brief scheme of steps to be taken in emergency management of an unconscious child is given below[8,37].

- Establish airway and give high flow oxygen by mask.
- Measure blood pressure and resuscitate with salt-containing fluids/ inotropes if low; do not reduce immediately if high.,
- Perform Dextrostix testing and simultaneous true blood sugar and give dextrose if low.
- Assess level of consciousness using the modified Glasgow coma scale (Table 1).
- Assess brainstem function (Table 4) and decide whether the patient has evidence of central or uncal herniation (Table 5).

- Lift the eyelids and look for tonic deviation of the eyes or nystagmus.
- Examine the fundi for papilledema (rarely seen in acute encephalopathy; absence does not exclude intracranial hypertension), retinal hemorrhages, and macular star suggestive of hypertension.
- If modified Glasgow coma score is less than 12 or there is evidence of herniation, intubate and ventilate.
- If modified Glasgow coma score is between 12 and 14, or. intubation is not possible immediately and there is evidence of progressive uncal or central herniation (Table 4), give mannitol 0.25 g/kg.
- If there is tonic deviation of the eyes or nystagmus, assume subtle status epilepticus and give a benzodiazepine and/or phenytoin.
- CT Brain should be done prior to lumbar puncture to rule out increased ICP and structural lesions.
- If the child is febrile and is either under the age of 12 months or is older than 12 months and has a Glasgow coma score greater than 12, undertake a lumbar puncture after checking that the child is not in subtle status. The CSF pressure should be measured with a transducer or a manometer. A dose of mannitol 0.25 g/kg should be given if the pressure is greater than 15 cm H_2O or if there is evidence of deterioration in the modified Glasgow coma score or the brain-stem signs after the lumbar puncture. If the CSF is cloudy, dexamethasone may be given before starting a third-generation cephalosporin.

- If the child is afebrile or febrile with a deteriorating level of conciousness, do not perform lumbar puncture, but start a third-generation cephalosporin and acyclovir[4,5].

ASSESSMENT

- History—through friend, family or emergency medical personnel
- General physical examination
- Neurological assessment—to define the nature of coma (Table 6.1).

The clinician has to determine:

- Where is the lesion responsible for coma?
- What is its nature?
- What is it doing?

The approach to clinical evaluation is used to categorize coma into:

- Coma without focal signs or meningism. This is the most common form of coma and results from anoxic, ischemic, metabolic, toxic, and drug- induced insults, infections, and post-ictal states.
- Coma without focal signs with meningism. This results from subarachnoid hemorrhage, meningitis, and meningoencephalitis.
- Coma with focal sings. This results from intracranial hemorrhage, infarction, tumor or abscess[26,28].

SPECIFIC NEUROLOGICAL EXAMINATION

1. Response to pain:

Painful stimuli should be administered without injury. This is done by pressure over the notch of the supraorbital nerve to induce a facial grimace, which will be present in the absence of limb responses with afferent peripheral lesions affecting the pain pathways. Limb response can also be assessed by pressing down on the nail bed with a tendon hammer or pinching the Achilles tendon .Asymmetry of response should be looked for as should the nature of the response, since this helps localise the site of structural damage.

2. Pupillary Responses

Assuming that the visual pathways up to the lateral geniculate body are intact, assessment of the pupillary responses is important in localising the site of lesion and separating structural from toxic metabolic causes, as pupillary responses in the latter are normal. Proper assessment of the pupillary responses requires a bright light and if needed magnification that can be provided by using an otoscope. Preceding ocular injury impairs responses and relatives should be asked about this. Use of mydriatics can confuse matters by causing an asymmetrical response as the effect may wear off asymmetrically. Drugs such as atropine or dopamine that can be used in resuscitation from cardiac arrest have effects on papillary reactions that may be misleading[17,18].

3. Ocular motility*:*

Centres for eye movement control are adjacent to the brainstem areas responsible for arousal; thus, EOM evaluation is a valuable guide to the presence and level of brainstem disease causing coma. Ocular pathways run from the midbrain to the pons, thus normal reflex eye movements imply that the pontomedullary junction to the level of the ocular motor nucleus in the midbrain is intact. In addition, the oculomotor nerve is susceptible to compression in tentorial herniation.

The following observations should be made:

- Resting position.
- Spontaneous eye movements.
- On lifting the lids and releasing them, observe tone and closure. If blinking is present, either spontaneously or to bright light, sound or menace, this implies an intact pontine reticular formation.
- When carrying out corneal reflexes, observe the movement of the eyelid and globe of the eye; with an intact pons eye closure will occur and with Integrity of both pons and midbrain, Bell's phenornenon will be present.
- Reflex eye movements should be performed[15,17].

4. Eye Deviation

This can be conjugate or disconjugate. Lateral deviation of the eyes is commonly caused by a lesion in the ipsilateral frontal eye fields, but can result from lesions anywhere in the pathway from frontal eye fields to the parapontine reticular formation (PPRF). Disconjugate eye movement imply sixth or third nerve or intrinsic brainstem lesions. Downward deviation of the eyes below the horizontal meridian is a sign of poor localising value, occurring in brainstem, bilateral thalamic, and subthalamic lesions and can occur in some metabolic encephalopathies. Upward deviation is also a poor localising sign being described both in sleep and seizures, as well as with brainstem lesions. Skew deviation occurs with posterior fossa lesions(13,21).

5. Spontaneous Eye Movements

If purposeful *eye* movements are present in an otherwise unresponsive patient, states confused with coma such as locked-in syndrome, catatonia, and pseudocoma should be considered.

Roving eye movements are slow, conjugate, lateral, to and fro excursions. These occur when third nerve nuclei are intact and often indicate a toxic, metabolic or alternatively bilateral hemisphere cause for coma. This can occur with or without other obvious manifestations of seizures, though sometimes these are simply subtle movements of eyelids,

tongue, jaw or face. The presence of such an eye movement disorder should raise the possibility of some form of complex partial status that should be confirmed by EEG.

Ocular bobbing describes a rapid downward jerk of both eyes with slow return to the mid-position. This eye movement disorder is specific for acute pontine lesions.

6. Motor Examination

Resting position and spontaneous movements should be documented. If the eyes and head are deviated to the side opposite hemiparesis, this implies a hemisphere lesion whereas deviation to the side of hemiparesis is indicative of a pontine lesion[11,21].

- Decerebrate rigidity—This refers to bilateral upper and lower limb extensor posture, usually the consequence of bilateral mid-brain or pontine lesions
- Decorticate posture—this refers to bilateral flexion of the upper limbs and extension of the lower limbs, usually the consequence of upper brainstem, lesion.

Unilateral decerebrate or decorticate postures can be seen and indicate a unilateral lesion. This asymmetry has some localising value.

7. Abnormal movements

Abnormal movements such as myoclonus, epilepsia partialis continua, and tonic-clonic seizures may all occur in coma. They are

important to identify since seizures require urgent treatment. Myoclonic jerking is seen commonly in patients with anoxic/ischemic encephalopathy and other toxic or metabolic disorders. Patients with brainstem herniation can have flexor or extensor posturing triggered by respiration or external stimuli. These should not be confused with seizures. Asymmetry of the plantar response, tendon jeerks, and muscle tone may all be valuable in localisation of structural lesions and differentiation from metabolic conidtions. Acute structural damage above the brainstem results in a flaccidity of muscle tone' and is asymmetric in comparison to metabolic disorders where such findings are usually symmetrical.

Table :4 Brainstem examination :

Response to pain	Flexion to supraocular pain extension to supraocular pain None	Diencephalic Midbrain/upper pontine Lower pontine
Posture	Normal Hemiparesis Decorticate Decerebrate Flaccid	Brainstem intact Uncal herniation Diencephalic Midbrain/upper pontine Lower pontine
Tone/reflexes/planters	Normal Unilateral pyramidal Bilateral Pyramidal Flaccid/extensor plantar	Brainstem intact Uncal herniation Diencephalic Lower pontine
Oculocephalic (Doll's) Exclude cord injury Turm head form side, watch eyes	Saccadic eye movements Full deviation eyes away Minimal deviation eyes No movement eyes	Normal forebrain control Diencephalic Midbrain / upper pontine Lower pontine.
Oculovestibular (calorics)	Nystagmus	Normal forebrain control

Exclude perforated eardum Head in midline and 30^0 back Inject 20ml ice cold into ear canal Pupil size	Full deviation eyes towards Minimal deviation eyes no movement eyes normal midpoint	Diencephalic Midbrain/upper pontine Lower pontine Midbrain/upper pontine.
	Small Uilaterally large Bilaterally large	Diencephalic Uncal hernlation Lower pontine
Pupil response to light Bright torch	Brisk Unresponsive	Brainstem intact Midbrain/upper pontine
Respiratory pattern	Normal Cheyene – Stokes Hyperventilation Ataxic, shallow Gasping, slow, irregular	Brainstem intact Diencephalic Midbrain / Upper pontine Lower pontine Medullary.

Role of Neurological Investigations

a. CSF Analysis

The only investigation that will confirm the diagnosis of CNS infections is lumbar puncture and CSF analysis. Lumbar puncture is contraindicated in the presence of gross papilloedema with or without neourological deficit or meningococcemia (risk of bleed). However LP has to be planned after a preliminary CT scan. Early CSF analysis may be normal and the lumbar puncture should be repeated if the meningeal signs persist. Send 3 bottles of CSF (8 drops or ½ CC in each bottle) for urgent gram stain and culture, virology study and biochemical study.

b. CT imaging is the most readily available investigation that gives immediate information on the presence of gross structural intracranial disease. This will confirm the presence of mass lesions showing displacement or shift of intracranial compartments—for example, subfalcine or uncal herniation. Raised intracranial pressure is suggested by narrowing of the third ventricle and loss of suprasellar cisterns. Magnetic resonance imaging provides better visualization of the brainstem and cerebellar structures, venous sinuses, compartment shifts and diffuse disorders-for example, laminar necrosis of hypoxic encephalopathy but in the acutely unwell or those who are ventilator dependent it is logistically difficult[24,26].

Neurologists should be aware of conditions where the CT brain scan is normal – for example, metabolic encephalopathies, but also disorders such as fat embolism.

c. The electroencephalogram (EEG) is helpful in the diagnosis of actue toxic or metabolic encephalopathies showing diffuse slow wave change (4-6Hz). Rapid (>12 Hz) acitivity occurs with sedative overdose and slow wave changes of a focal nature are found in herpes simplex encephalitis. αComa-where the normal cortically generated α rhythm is retained – occurs in hypoxic ischemic or drug induced states. This alpha activity is uninfluenced by stimulation or eye opening (in an alert awake patient alpha rhythm disappears with eye opening) and this suggests a

better prognosis. Apart from this the EEG is not as good a predictor of clinical outcome when compared with clinical assessment. However, the EEG is of particular value in confirming complex partial status, a condition that should always be considered in the intensive care setting in patients with an ischemic hypoxic insult and low coma score[2,4,5].

d. MRI provides best visualization of the posterior cranial fossa and its contents, an extremely useful feature when structural lesion of the brainstem, viralencephalitis and demyelination are suspected. At present MRI is limited by length of time to perform and image degradation by even a slight movement of the patients.

Table :5. Herniation syndromes

Uncal	Unilateral fixed dilated pupil Unilateral ptosis Minimal deviation of eyes on oculocephalic / oculovestibular testing Hemiparesis
Diencephalic	Small or midpoint pupils reactive to light full deviation of eyes on oculocephalic / oculovestibular testing Flexor response to pain and / or decorticate posturing. Hypertonia and / or hyperreflexia with extensor plantars. Cheyne – Stokes respiration.
Midbrain / upper pontine	Midpoint pupils, fixed to light Minimal deviation of eyes on oculocephalic / oculovestibular testing Extensor response to pain and / or decerebrate posturing Hyperventilation.
Lower pontine	Midpoint pupils, fixed to light No response on oculocephalic / oculovestibular testing. No response to pain or flexion of legs only Flaccidity with extensor plantars shallow or ataxic respiration.
Medullary	Pupils dilated and fixed to light slow, irregular, or gasping respiration Respiratory arrest with adequate cardiac output

Related studies

Indian study[7]: (Bansal et al) was done on the etiology and clinical profile of non-traumatic coma in children and to determine the clinical signs predictive of outcomeEtiology of coma in 60% cases was CNS infection (tubercular meningitis-19, encephalitis-18, bacterial meningitis-16, others-7); other causes were toxic-metabolic conditions (19%), status epilepticus (10%), intracranial bleed (7%), and miscellaneous (4%). 65 children survived, 11 were normal, 14 had mild disability, 21 had moderate disability and 14 were severely disabled and dependent. Survival was significantly better in patients with CNS infection (63%) as compared to those with toxic-metabolic causes (27%) and intracranial bleed (43%, $P < 0.05$) CNS infections were the most common cause of non-traumatic coma in childhood. Simple clinical signs were good predictors of outcome.

Malaysia study:

Sofiah A et al was done on[36]

Childhood non- traumatic coma in Kuala Lumpur,

All post – neonatal children with acute non-traumatic coma admitted over an 8-month period were analysed and followed up for 18-24months to determine the aetiology and outcome of their coma. One hundred and sixteen children, 72boys and 44girls were recruited. Half the children were under 1 years of age only 16(14%) were more than 6 years

of age. Eighty cases (69%) were due to infection, 15(13%) to toxic metabolic causes, six (5%) to hypoxic ischaemic insults, four (3.5%) had intracranial haemorrhage, nine (7.8%) were due to miscellaneous causes and in two (1.7%) the cause was unknown. Age of onset and sex did not significantly affect outcome.

Saudi Arabia study[18]

A.M Ali[1] et al was done study on Traumatic and non-traumatic coma in children in the referral hospital, Al-Hasa, Saudi Arabia

Determined the incidence, etiology and outcome of paediatric coma patients in King Fahad Hospital, which is the only referral centre for Al-Hasa region, Saudi Arabia. From April 1999 to March 2002, 91 children with coma (age range 28 days to 12 years) were admitted. The Glasgow Coma Scale for children was used for assessment. Neurological outcomes were categorized as intact, impairment or death. Incidence of coma was 4.77 per 100 000 population per year. Trauma (head trauma or polytrauma) was the commonest cause of coma (52.8%), followed by infection (25.3%). Mortality was 47.2% (35.4% among traumatic cases and 60.5% among non-traumatic cases). There was impaired outcome in 19.8% of patients (22.9% with traumatic coma and 16.3% with non-traumatic coma).

Nigeria study[79]

Ogunmekan, et al done hospital based study on non-traumatic coma in childhood: etiology, clinical findings, morbidity, prognosis and mortality were reviewed The records of 225 comatose children aged 6 weeks to 10 years seen in the Children's Emergency Room of the Lagos University Teaching Hospital were studied.

Traumatic cases were excluded. Etiology, clinical findings, systemic disturbances, and factors predicting outcome were correlated. Intracranial infection (n = 94) was the most frequent cause of the coma. Associated grand mal seizures were reported in 152 children (68 per cent), most frequently in those aged 1 to 3 years. Metabolic acidosis or other systemic disturbances, usually multiple, occurred in nearly half the children. Sixty children died. Ocular and motor abnormalities correlated significantly with mortality. At discharge, 102 of the remaining 165 children were normal and 63 had mild to severe handicap.

Egypt[12]

Hala fouad et al did the prospective descriptive study of 100 consecutive pediatric nontraumatic coma cases to identify etiology, clinical profile, and predictive outcome in a pediatric emergency department at a tertiary care university hospital. Most frequent etiologies were metabolic (33%), central nervous system infections (28%), and

intracranial hemorrhage (13%). In the emergency department, 50% of those patients died. Hypothermia, hypotension, flaccidity, and poor Glasgow coma scale at admission correlated significantly with mortality. After 48 hours of admission, poor pulse volume, poor Glasgow coma scale, abnormal respiratory pattern/apnea, and seizures correlated significantly with mortality. On logistic regression, poor Glasgow coma scale at admission, abnormal respiratory pattern, and seizures after 48 hours of admission were independent significant predictors of mortality. Metabolic causes are the most common etiology in pediatrics nontraumatic coma. Simple clinical signs were good predictors of outcome.

Iran study[67]

Fariba et al did a retrospective cross sectional study over a perid of 5 years. Files of 150 children aged between 1 month and 14 years admitted with non-traumatic coma to pediatric intensive care unit of Rasool Akram hospital were reviewed. Etiology of coma in 49 patients (32.7%) was infectious (meningitis, encephalitis, respiratory and systemic). Other causes were status epilepticus 44(29.4%) metabolic (drowning, electrical shock, suffocation)9 (6%) shunt dysfunction (secondary to congenital brain malformations) 7 (4.6%) other (acute disseminated encephalomyelitis, vasculitis, hypertensive encephalopathy)

11 (7.3%) unknown 9(6%) Infection occurred significantly (p=0.002)in those between 2 and 6 years. Overall 25 children (16.6%) died. Of those survived 16 became severely disable. Accidents and infections had higher mortality compared to other groups (p<0.001 and P=0.02 respectively).

United kingdom (UK) study[24]

C.P Wong, et al did population based study on incidence, aetiology, and outcome of non-traumatic coma which revealed that the incidence of non-traumatic coma was 30.8 per 100 000 children under 16year (6.0 Per 100 000 general population per year). The age specific incidence was notably higher in the first year of life (160 per 100 000 Children per year). CNS specific presentations became commoner with increasing age. In infants, nearly two thirds of presentations were with non-specific, systemic signs. Infection was the commonest overall aetiology, Aetiology remained unknown in 14% despite extensive investigation and/or autopsy. Mortality was highly dependent on aetiology, with aetiology specific mortality rates varying from 3% to 84% with follow up to approximately 12 months, overall series mortality was 46%.

AIMS OF THE STUDY

Aim:

(i) To study the etiology of the Impairment of consciousness and Coma in paediatric population in South Tamilnadu.

(ii) To study the outcome of Impairment of Consciousness and Coma due to various etiologies and the factors influencing the outcome in pediatric population in South Tamilnadu.

EVALUATION AND OUTCOME OF IMPAIRMENT OF CONSCIOUSNESS AND COMA IN PAEDIATRIC POPULATION

Materials and methods:

The study population was selected from the Paediatric Intensive Medical Care Unit, Department of Paediatrics, Tirunelveli Medical College Hospital, a tertiary care centre. One hundred patients were selected for study in the age group ranging from 01 month to 12 years over a period of 10 months from March 2010 to December 2010.

Selection of Patients:

Patients admitted in the clinical spectrum of various stages of continuum of coma from state of minimal reduction of wakefulness and arousable drowsiness to unarousable unresponsiveness of coma for more than duration of one hour (Ref: Pediatric Neurology Swaiman, IV edition Page 1378).

Exclusion criteria

- Patients less than one month of age.
- Traumatic brain injury patients
- Patients with previous mental and physical disability

Ethics:

This study was approved by Institutional Ethics committee.

Methods:

This study was conducted as hospital based prospective observation study. History, clinical findings and laboratory data were collected. In assessing effects of age on etiology and outcome, study population was divided into three age bands, as infants and toddlers - o1 month – 3years, preschool children 4-5 years and school going children 6-12 years. The mean age in months was 57.0± 35.9. The range of age was 3months to144 months. Sex of patients was registered. Presenting symptoms were assigned to one of three symptom group as constitutional non specific such as fever, nausea, vomiting, incessant cry, poor activity and refusal to feed, and CNS related non-focal-irritable, drowsy, headache, neck pain, confusion and focal. The clinical findings recorded were vital parameters like temperature, pulse, respiratory rate, heart rate, blood pressure and CNS non-focal signs as meningeal irritation and focal signs as brain stem reflexes, pupillary size, shape and light reflex, Doll's Eye movement , respiratory pattern, motor pattern presence and absence of seizures and types of seizures were noted. Conscious status qualitatively assessed as irritable, drowsy but arousable and coma in all age group of children and quantitatively assessed by using Glasgow coma scale scores for the age group of 6-12 years. In those less than 5 years of age modified GCS was used. Duration of impairment of consciousness

and coma was observed and noted as $\bar{<}$ 1day $\bar{<}$3days $\bar{<}$7days, $\bar{<}$14days and > 14 days at time of the discharge or death at the hospital.

Data of laboratory investigations collected as systemic basic and routine, CNS related – CSF analysis culture and sensitivity and neuroimaging modalities as CT and MRI Brain.

Etiology was classified into Acute CNS infections, Epilepsy, Hypoxic Ischemic encephalopathies, Metabolic, Toxic and Others.

Description of Etiology categories

CNS infection:-

Diffuse or multifocal or focal, unilateral or bilateral infection of the brain due to bacteria, virus, fungus and parasites.

Epilepsy:

Epilepsy is defined as a condition of recurrent unprovoked seizures. An epileptic seizure (fit) is the manifestation of an abnormal and hypersynchronous discharge of a set of cerebral neurons manifesting as sudden and transient motor, psychic or sensory phenomena, with or without alteration in awareness. Status epilepticus is internationally classified as a seizure lasting for more than 30 minutes or recurrent seizures lasting for more than 30 minutes from which the patient does not regain consciousness [ILAE , 1981]

Hypoxic Ischemic encephalopathy:

HIE is due to cardiorespiratory circulatory insufficiency because of infective and non-infective causes of respiratory, cardiac, heamotological and other disorders.

Metabolic Encephalopathy

IC/C is due to inborn error of metabolism and acquired causes of hepatic, renal, endocrine and electrolyte disorders.

Intoxication:

IC/C is due to overdose of drugs, toxins, poisonous animal bites and stings.

Others:

IC/C is due to vascular, autoimmune, malignancy and rare diseases.

The primary etiology of impairment of consciousness and coma was determined after reliable history, focussed clinical examination and relevant laboratory investigations. The following clinical variables such as age, sex, age specific etiology, severity of IC/C by GCS/MGCS, duration of IC/C, signs of meningeal irritation, focal deficit, presence or absence of seizures with specific etiology and CT Brain results were studied to find out the correlation of etiology and impact upon the outcome of IC/C.

Treatment :

The admitted cases were treated by a team of pediatrician, neurologist and other specialists.

Pediatric Advanced Life Support (PALS) strategies and therapeutic guidelines were followed as recommended by IAP(Indian Academy of Pediatrics) and AAP (American Academy of Pediatrics)

Outcome:

Neurological outcome was broadly divided into grades I-Good recovery-Patients who regain the ability to conduct a normal life or, if a preexisting disability exists, to resume the previous level of activity. II - Moderate disability- Patients who achieve independence in daily living but retain either physical or mental limitations that preclude resuming their previous level of function. III- Servere disability – Patients who regain at least some congnitive function but depend on others for daily support. IV - Vegetative state-Patients who awaken but give no sign of cognitive awareness. V-No recovery-Patients who remain in coma until death .Serial observations was done at 24 hours, 3^{rd} 7^{th}, 14^{th} day and at the time of discharge by presence or absence of clinical signs that reflect the extent and severity of dysfunction of cerebral hemispheres and brainstem and outcome was determined.

Statistical analysis:

The statistical procedures were performed by software namely statistical products and service solutions (S.P.S.S.13.0). The non-metric data of clinical variables and associations were tested by the test of significance, Chi-squared Test. The metric data was tested by students 't' test. P value less than 0.05 were considered as significant ($P < 0.05$).

Observation, Analysis and Results

Table :1

Etiology by occurrence

CNS Infection	38 % (38)
Epilepsy	21 % (21)
Hypoxic / Ischemic	23% (23)
Toxic	6% (6)
Metabolic	8% (8)
Others	4% (4)

Table 1 results shows decending order of occurrence as Acute CNS infection, Hypoxic ischemic encephalopathy, Epilepsy, Metabolic, toxic and others in over all pediatric population.

Acute CNS infection is the commonest cause of impairment of consciousness and coma and equally second commonest causes are hypoxic – ischemic encephalopathy and Epilepsy.

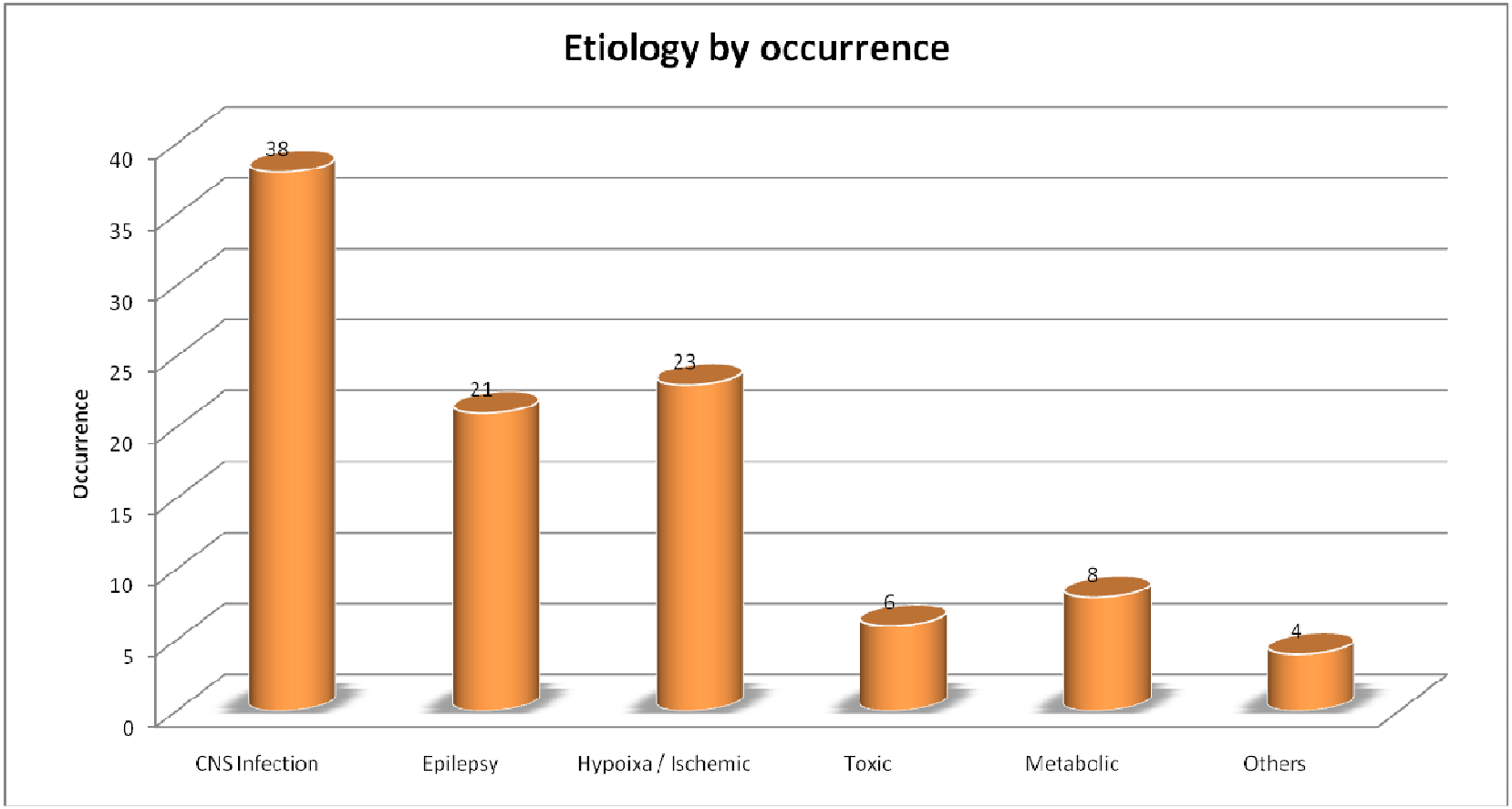

Etiology by occurrence
Occurrence
0
5
10
15
20
25
30
35
40
38
21
23
6
8
4
CNS Infection
Epilepsy
Hypoixa / Ischemic
Toxic
Metabolic
Others

Table:2

Age by occurrence

1month-3 Years	48% (48)
4-5 years	32% (32)
6-12 years	20% (20)

1.Table 2 results shows age groups. Most susceptible age group was o1 month – 3years, second 4-5 years and least was 6-12 years.

Very lower extreme age groups were more vulnerable for diseases causing of impairment of consciousness and coma in pediatric population.

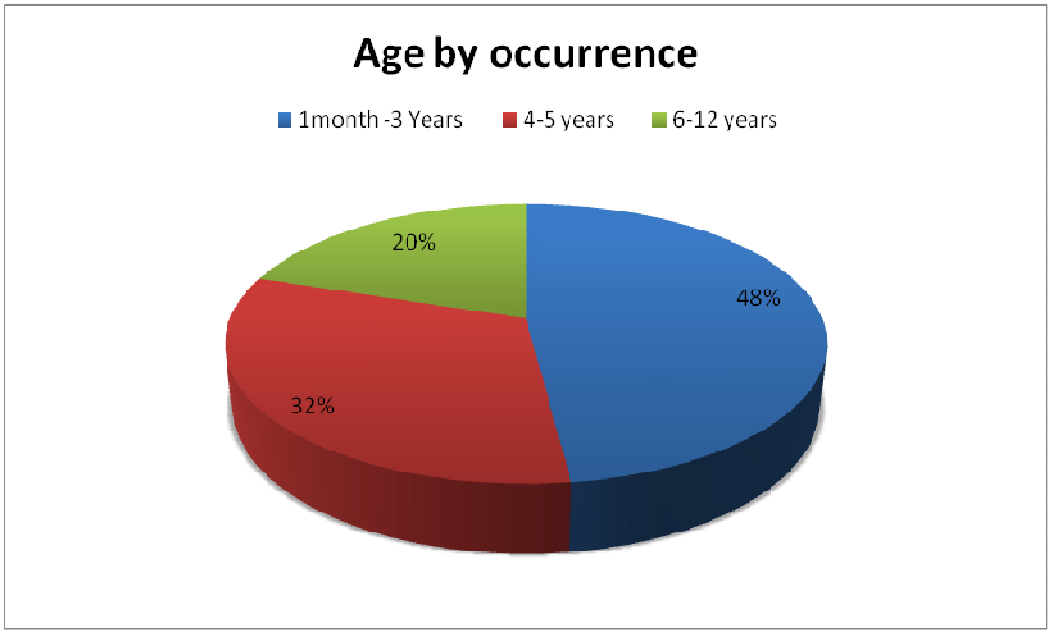


Table:3

3.Age specific etiology:

Age and Etiology	Infection	Epilepsy	Hypoxic Ischemic	Metabolic	Toxic	Others
01month-3years (48) years	35.4% (17)	14.5% (7)	41.6% (20)	4.16% (2)	2.08% (1)	2.08% (1)
4-5 years (32)	34.3% (11)	31.2% (10)	9.37% (3)	12.5% (4)	12.5% (4)	0
6-12 years (20)	50% (10)	20% (4)	(0)	10% (2)	5% (1)	15% (3)

Table 3 shows common etiology occurrence within specific age groups. Among 01 month 3-years hypoxic ischemic Encephalopathy and acute CNS Infection were commonest etiology and among 4-5 years both infection and epilepsy were commonest etiology. In overall pediatric population CNS infection is the leading cause of impairment of consciousness and coma. The association between the age and specific etiology was statically significant ($p< 0.05$).

In the approach of Impairment of consciousness and coma in children, age – specific etiology should be thought of.

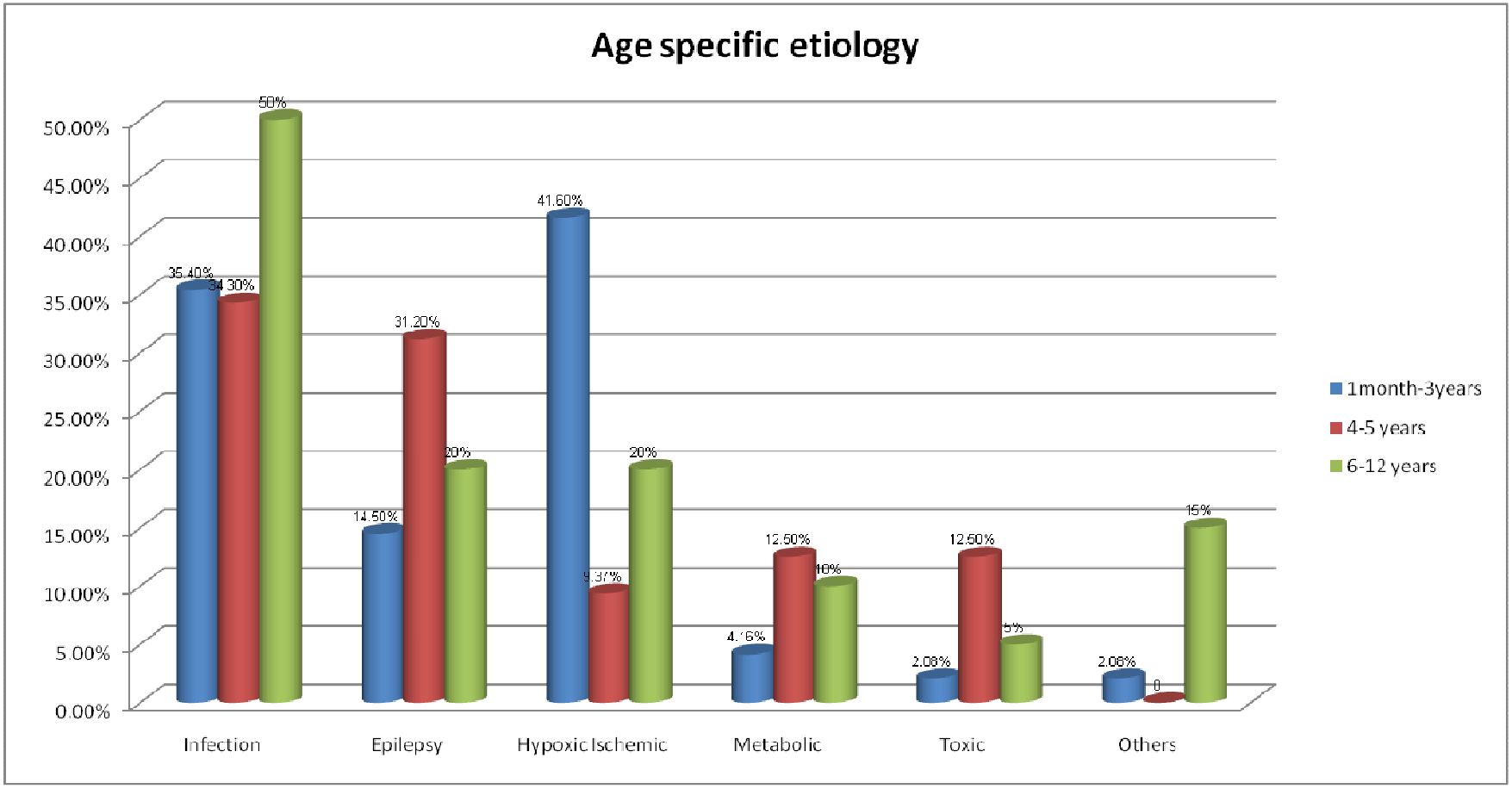
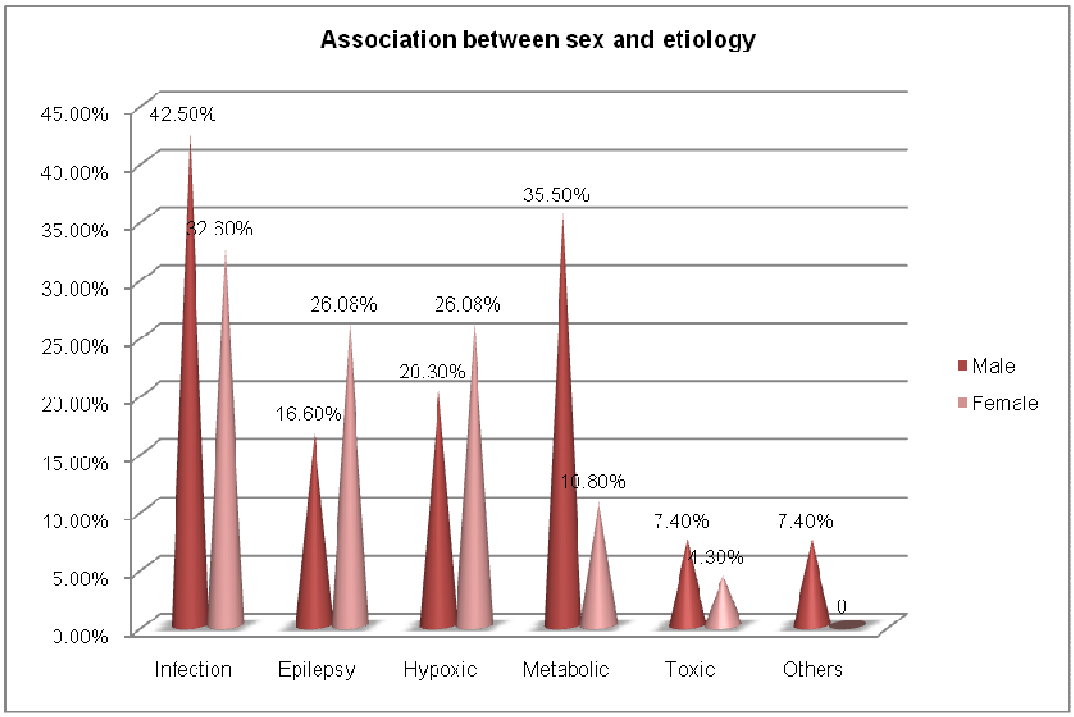

Age specific etiology
50.00%
45.00%
40.00%
35.00%
30.00%
25.00%
20.00%
15.00%
10.00%
5.00%
0.00%
50%
35.40%
34.30%
31.20%
20%
14.50%
41.60%
20%
12.50%
10%
12.50%
2.06%
2.06%
0
15%
Infection
Epilepsy
Hypoxic Ischemic
Metabolic
Toxic
Others
1month-3years
4-5 years
6-12 years

Table:4

4. Association between sex and etiology:

Sex	Total	Infection	Epilepsy	Hypoxic	Metabolic	Toxic	Others
Male	54	42.5% (23)	16.6% (9)	20.3% (11)	5.55% (3)	7.4% (4)	7.4% (4)
Female	46	32.6% (15)	26.08% (12)	26.08% (12)	10.8% (5)	4.3% (2)	0
Total	100	38	21	23	8	6	4

Table 4 shows sex distribution of IC/C in children and slight preponderance of male gender but it is not statistically significant when comparing the relationship of sex with etiology. CNS infection was the commonest in both sex. In overall observation the association of sex and etiology were statistically not significant (P>0.05).



5. Presenting Complaints

Non specific	Fever	62%
	Nausea, vomiting	84%
	Refusal to feed	90%
	Incessant cry	23%
	Poor Activity	96%
CNS Complaints		
	Drowsy	72%
	Irritable	36%
	Headache	70%
	Seizures	70%
	Confusion irrelevant talk	42%
	Neck Pain	28%
	Focal weakness	9%

5. Table 5 shows common and not uncommon presentation of non-specific constitutional symptoms as poor activity, refusal to feed, fever, incessant cry and CNS related symptoms as drowsy, irritable, headache, neck pain, seizures.

If constellation of above mentioned symptoms are present in a child the treating pediatrician should recognize them as red flag symptoms of IC/C.

Table :6

6. Association between meningeal and focal signs and etiology (F & M).

Etiology							
Signs	**Infection**	**Epilepsy**	**Hypoxic**	**Metabolic**	**Toxic**	**Others**	**Total**
Normal	19.1% (13)	30.8% (21)	26.4% (18)	11.7% (8)	8.8% (6)	2.9% (2)	68
F & M	78.1% (25)	0	15.6% (5)	0	0	6.2% (2)	32
Total	38	21	23	8	6	4	100

The above table 6 describes the meningeal and focal signs of the study subsects. 32% of patients showed them and 68% did not. The above signs were strongly associated with infection. The other etiologies were associated with normal subsects. The above associations were statistically very highly significant. (P<0.001)

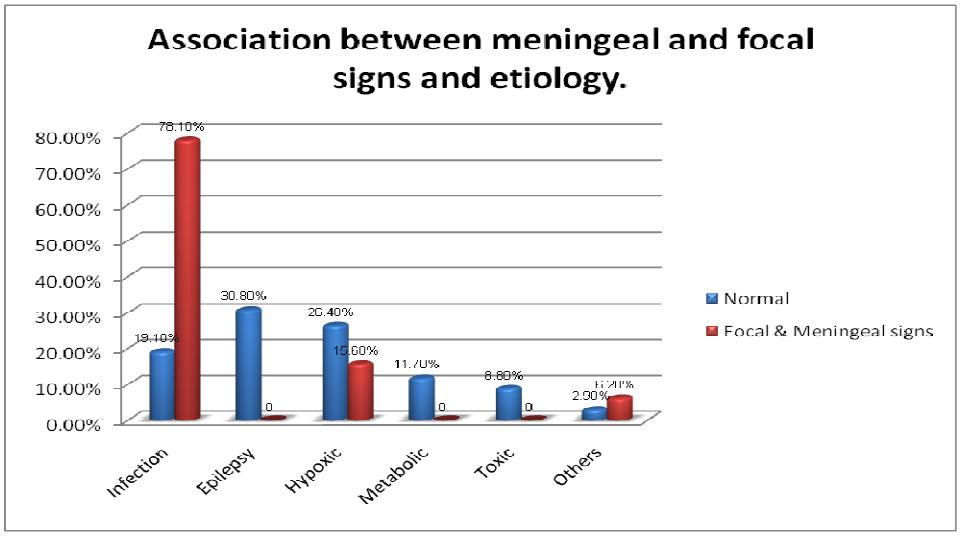


Table : 7

7. Comparison of association between the disease related seizures and non-epileptic etiological patients.

	Etiology						
	Infection	**Hypoxic**	**Metabolic**	**Toxic**	**Others**	**Total**	**%**
Disease related seizures	70% (35)	22% (11)	0	2% (1)	6% (3)	50	63.3
Normal	10.3% (3)	41.3% (12)	27.5% (8)	17.2% (5)	3.44% (1)	29	36.7
Total	38	23	8	6	4	79	100.0

In the above Table 9 among the non-epileptic etiological patients (79) disease related seizures were present in 63.3% and normal was 36.7%. Disease related seizures were associated with CNS Infection predominantly. Metabolic and toxic disease rarely presented with seizures. The above associations were statistically very highly significant ($P<0.001$).

Table : 8

8.Comparison of association between duration of non-traumatic IC/C with etiology.

Etiology							
Duration (days)	**Infection**	**Epilepsy**	**Hypoxic**	**Metabolic**	**Toxic**	**Others**	**Total %**
≤ 1	0	41.6% (10)	50% (12)	0	4.16% 1	4.1% 1	24
> 1 to 3	28.1% (9)	18.5% (6)	12.5% (4)	18.7% (6)	15.62% (5)	6.2% (2)	32
4 to 7	57.1% (20)	14.28% (5)	20.1% (7)	5.7% (2)	0	2.8% 1	35
7 to 14	100% (6)	0	0	0	0	0	6
>14	100% (3)	0	0	0	0	0	3
Total	38	21	23	8	6	4	100

The above table 10 describes the duration of days with etiology. The duration of days. <1 day 24%, <3 days 32%, <7 days was 35%, <14 days was 6% and >14 days was 3%. The one day duration was mostly associated with hypoxic 41.6 and epilepsy 50%, 3days duration was associated with infection 28.1 epilepsy 18.7, metabolic 18.7, and 7to14 and more than 14days were highly associated with infection.

The above associations between the duration and etiologies were statistically very highly significant (P<0.001).

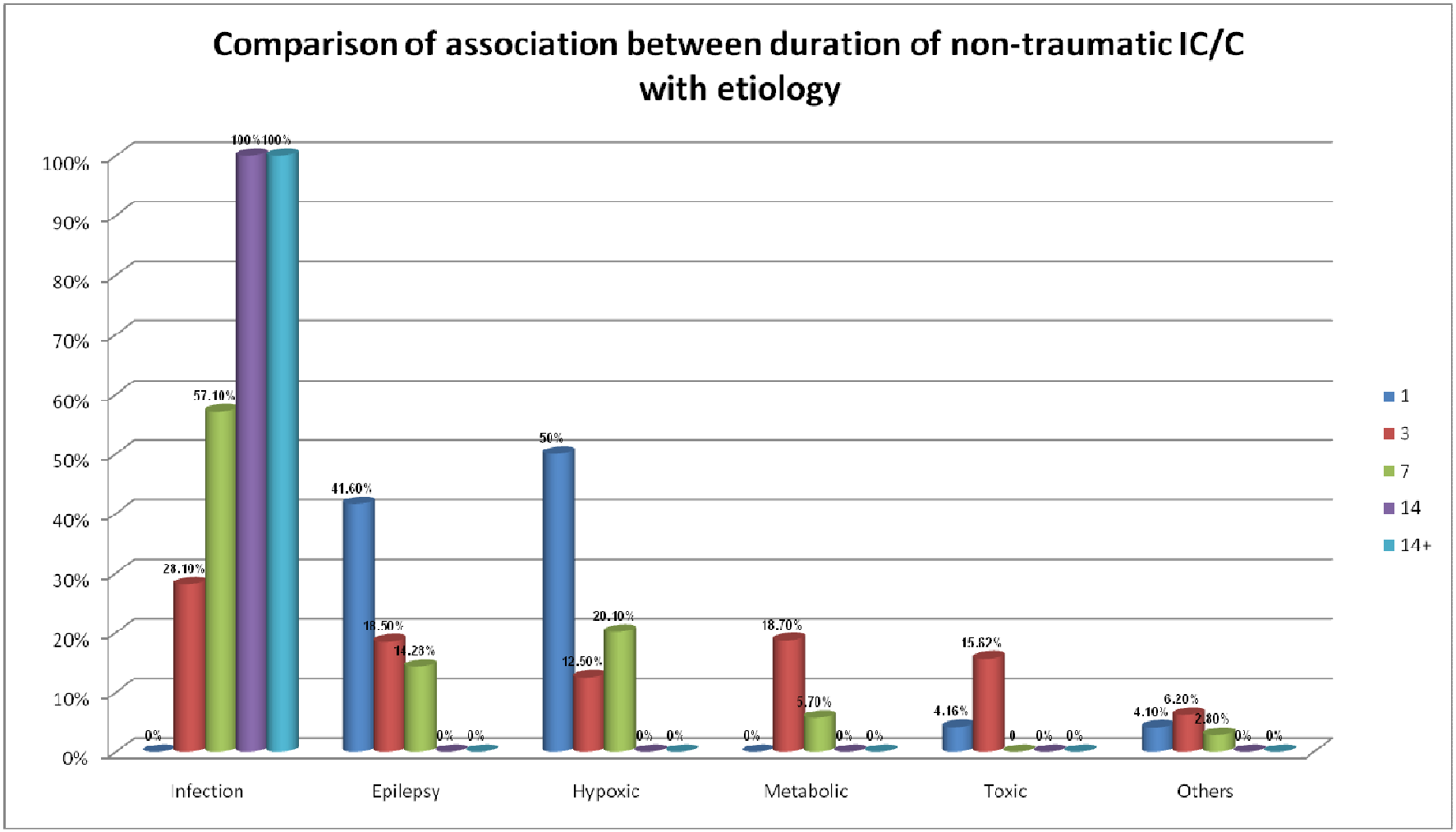

Comparison of association between duration of non-traumatic IC/C with etiology
100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
0%
28.10%
57.10%
100%
100%
41.60%
18.50%
14.23%
0%
0%
50%
12.50%
20.10%
0%
0%
0%
18.70%
5.70%
0%
0%
4.16%
15.62%
0
0%
0%
4.10%
6.20%
2.80%
0%
0%
Infection
Epilepsy
Hypoxic
Metabolic
Toxic
Others
1
3
7
14
14+

Table : 9

9.Association between the CT Brain results with etiology.

Etiology								
CT Brain results	**Infection**	**Epilepsy**	**Hypoxic**	**Metabolic**	**Toxic**	**Others**	**Total**	**%**
Positive	91.3% (21)	4.3% (1)	0	0	0	4.3% (1)	23	36.5
Negative	42.5% (17)	42.5% (17)	7.5% (3)	2.5% (1)	0	5% (2)	40	63.5
Total	38	18	3	1	0	3	63	100

Among the two subsects 63 had undergone CT brain imaging. Among the CT Brain subsects 63.5% were normal and the remaining 36.5% were positive. The positive cases were associated with the infection (91.3%) and the remaining negative cases were associated with other etiologies. The above associations were statistically very highly significant ($P<0.001$).

Outcome of Impairment of Consciousness and Coma in Pediatric Population :

Table :1

Association between the Etiology and Outcome

	I	II	III	IV	V
Infection (38)	68.4% (26)	9.09% (3)	2.6% (1)	10.5% (4)	10.5% (4)
Epilepsy (21)	66.6% (14)	19.04% (4)	14.2% (3)	0	0
Hypoxia / Ischemic (23)	60.8 (14)	8.6 (2)	0	0	30.4% (7)
Metabolic (8)	75% (6)	0	0	0	25% (2)
Toxin(6)	100%(6)	0	0	0	0
Others(4)	25%(1)	50%(2)	0	0	25%(1)

*Numbers in Parenthesis

Table 1 shows. Etiological relationship with outcome in tertiary care centre. Hypoxic ischemic encephalopathy cases had poor outcome and grade V outcome was more in number than CNS infections, seizures. Infective etiology had all types of outcome. Grade I was more in both infective and seizure etiology. Metabolic, toxic and other cases are very small in number and outcome study needs large number of cases to conclude. The association between the etiology and outcome was statistically significant ($P < 0.05$).

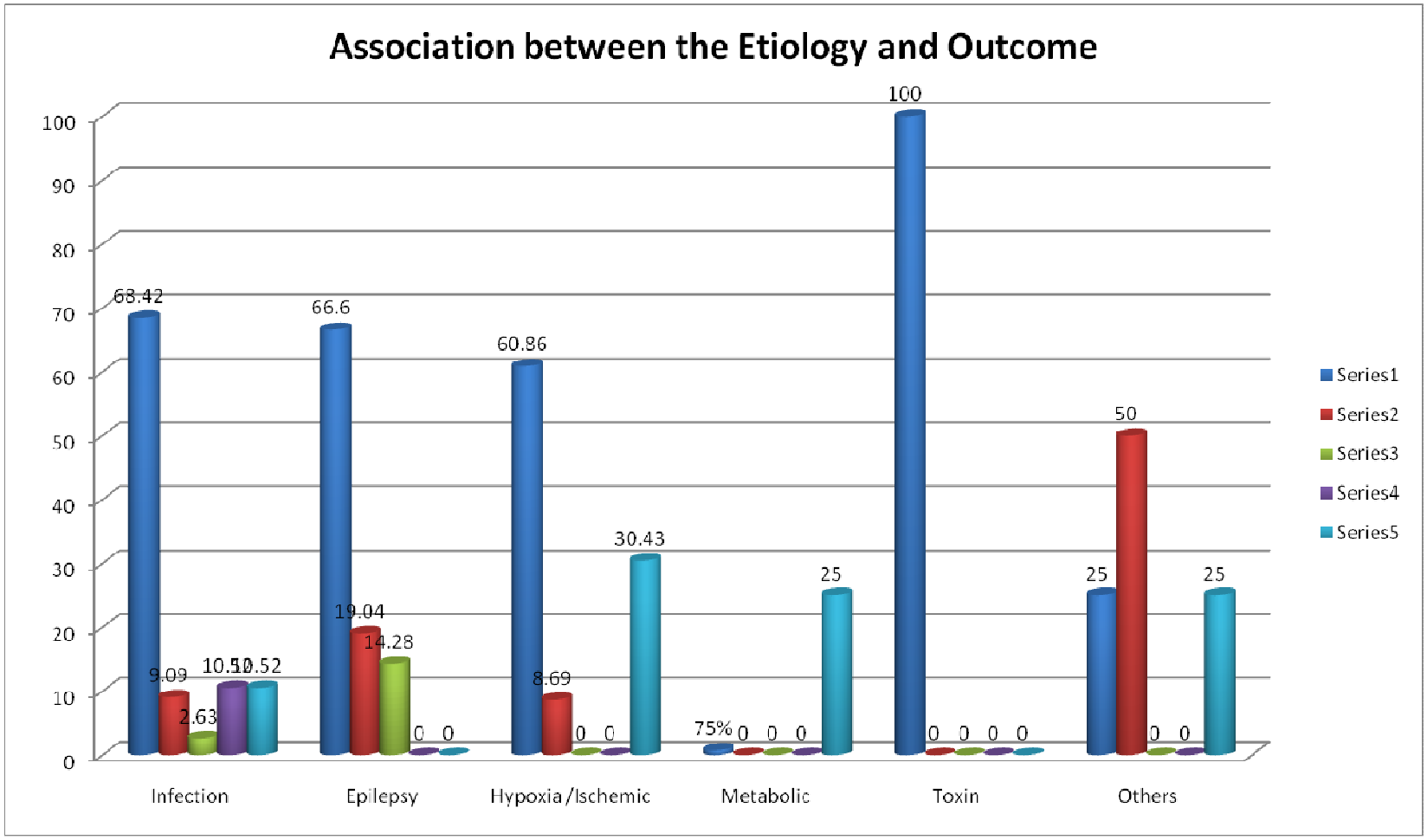
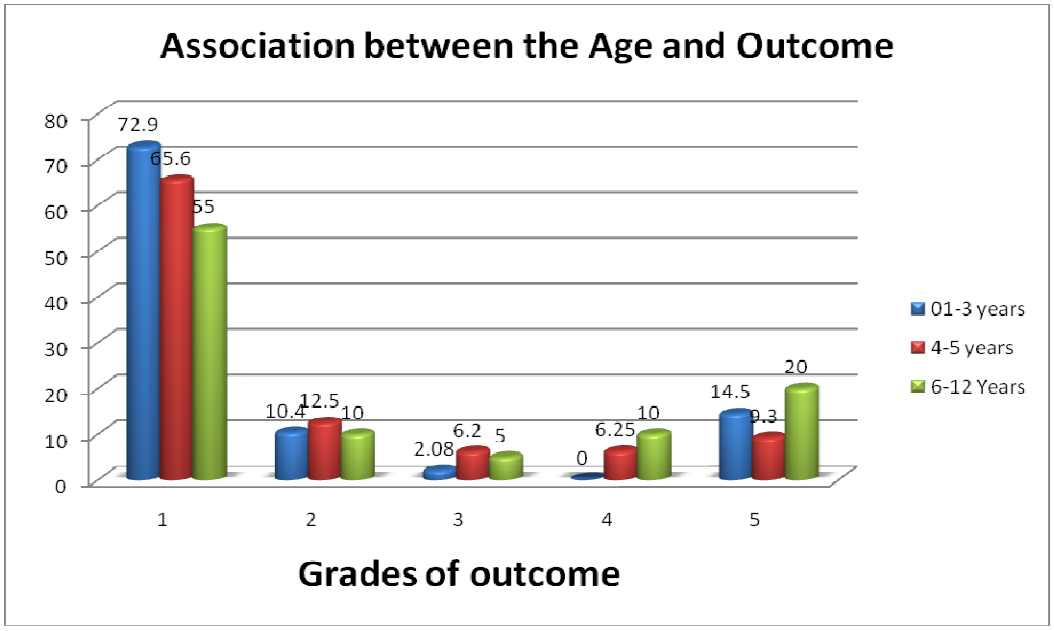

Association between the Etiology and Outcome
100
90
80
70
60
50
40
30
20
10
0
68.42
9.09
2.63
10.52
66.6
19.04
14.28
0
0
60.86
8.69
0
0
30.43
75%
0
0
0
25
100
0
0
0
0
25
50
0
0
25
Infection
Epilepsy
Hypoxia/Ischemic
Metabolic
Toxin
Others
Series1
Series2
Series3
Series4
Series5

Table :2

2. Association between the Age and Outcome

	I	II	III	IV	V
01-3 years(48)	72.9%(35)	10.4%(5)	2.08%(1)	0	14.5%(7)
4-5 years(32)	65.6%(21)	12.5%(4)	6.2%(2)	6.25%(2)	9.3%(3)
6-12 Years(20)	55%(11)	10%(2)	5%(1)	10%(2)	20%(4)

Table 2 shows age criteria is not a determining factor to conclude Outcome. Outcome did not depend upon age factor due to heterogenous nature of etiology in impairment of consciousness and coma. No significant associations were prevailing between the age and outcome (P>0.05).



3. Association between sex and outcome

	I	II	III	IV	V
Male(53)	67.2%(36)	11.32% (6)	3.77% (2)	1.88% (1)	15.09% (8)
Female(47)	65.95%(31)	10.6% (5)	4.25% (2)	6.38% (3)	12.76% (6)

Table 3 shows slight preponderance of male in case of impairment of consciousness and coma and it did not have statically significance in outcome. Sex was not dependable criteria for outcome. The above association were statistically not significant (P>0.05).

4.Association between age specific common etiology and outcome

Age1month-3years	I	II	III	IV	V
Infection(17)	76.47%(13)	5.88%(1)	5.88%(1)	0%	11.76%(2)
Epilepsy(7)	85.71%(6)	14.24%(1)	0%	0%	0%
Hypoxic and Ischemic(20)	65%(13)	10%(2)	0%	0%	25%(5)

Age4-5years	I	II	III	IV	V
Infection(11)	63.36%(7)	9.09%(1)	0%	18.18%(2)	9.09%(1)
Epilepsy(10)	50%(5)	30%(3)	29%(2)	0%	0%

Age 6-12 years	I	II	III	IV	V
Infection(10)	60%(6)	10%(11)	0%	20%(2)	10%(1)
Epilepsy (4)	75%(3)	0%	25%(1)	0%	0%

4. Table 4 shows relationship of age specific common etiology and outcome. Hypoxic ischemic encephalopathy in 01month-3year age group patients had poor prognosis compared with infective and epilepsy etiology. Infective etiology in 4-5 year are 6-12 year age group patients had good prognosis compared with other etiological group of patients in the same age band. The associations were statistically significantly ($P<0.025$).

5.Association between GCS/MGCS and outcome.

Grades of outcome						
GCS	I	II	III	IV	V	Total
8	50% (11)	0	9%(2)	9%(2)	31.8%(7)	22
10	0	0	0	50% (2)	50%(2)	4
11	50% (1)	50%(1)	0	0	0	2
12	77.7%(21)	7.4%(2)	0	0	14.8%(4)	27
13	75.5%(34)	17.7%(8)	4.4%(2)	0	2.2%(1)	45
Total	67	11	4	4	14	100

The association between the GCS score with grades of outcome was analysed and interpreted in the above table5. The results showed that there was a significant associations between the score 13 with grade I and II. The low score 8 was associated with the grade V. The above associations were statistically very highly significant ($P<0.001$).

6. Association between duration of IC / C and outcome

	I	II	III	IV	V	Total
≤ 1day	91.6% (22)	4.1% (1)	0	0	4.1% (1)	24
> 1 to 3 Days	65.6% (21)	12.5% (4)	9.3% (3)	0	12.5% (4)	32
4 to 7 days	62.8% (22)	14.2% (5)	2.8% (1)	2.8% (1)	17.1% (6)	35
7 to 14 days	33.3% (2)	16.6% (1)	0	33.3% (2)	16.6% (1)	6
>14 days	0	0	0	33.3% (1)	66.6% (2)	3
Total	67	11	4	4	14	100

6. Table 6 shows shorter duration of impairment of consciousness coma got better outcome and those children who had prolonged duration of impairment of consciousness had poor outcome. Duration of Impairment of consciousness is an independent factor to determine the outcome. The association was statistically very highly significant ($P<0.001$).

7. Association between presentation with and without focal signs and meninglsm and outcome

	I	II	III	IV	V	Total
With focal signs and meningism	59.3% (19)	15.6% (5)	0	12.5% (4)	12.5% (4)	32
Without focal signs and meningism	70.5% (48)	8.8% (6)	5.8% (4)	0	1.4% (10)	68

Table 7 shows pt with focal signs and meningism had poor outcome compared with who had no focal signs and menigism. Comparatively grade I outcome was more and Grade V outcome was lesser in patients who had no focal signs and meningism than who have it. The above association were statistically significant ($P<0.05$)

8.Association between presentation with and without disease related seizures and outcome

Grades of outcome						
	1	2	3	4	5	Total
With Seizures	66% (33)	6% (3)	2% (1)	8% (4)	18% (9)	63.2% (50)
With out Seizure	68.9% (20)	2% (4)	0	0	17.2% (5)	36.8% (29)
Total	53	7	1	4	14	79

8. Table 8 Shows that patients with secondary seizures with specific etiology in context of impairment of consciousness had poor outcome than those who had no secondary seizures. So occurrence of multiple or prolonged seizures in impairment of consciousness patients have unfavorable effect. Aggressive control of seizures is mandatory to get better outcome.

9.Association between CT brain results and outcome

Grade of outcome							
CT brain results	I	II	III	IV	V	Total	%
Positive	73.9% (17)	13.04% (3)	4.3% (1)	4.3% (1)	4.3 % (1)	23	36.5
Normal	67.5% (27)	7.5% (3)	7.5% (3)	7.5% (3)	10% (4)	40	63.5

9. Table 10 shows that CT Brain results either + or – in IC/C children, did not have major impact upon outcome. But this needs to be confirmed by larger studies.

DISCUSSION

Consciousness refers to the state of awareness of self and environment. Evaluation of consciousness in pediatric patient must take into account age and the appropriate developmental level. The diagnosis of coma and impairment of consciousness involves both state and reactivity

Clouding of consciousness is the minimal reduction of wakefulness or awareness where in the main difficulty is attention or vigilance. Confusion is the state of impaired ability at developmentally and intellectually appropriate level.

Several altered state of consciousness with activated mental state can be seen in older children and may be difficult to differentiate from each other. Coma usually requires a period of unconsciousness for at least 1 hour to distinguish from syncope, concussion or other states of transient unconsciousness.

The history, physical examination, laboratory and other investigation are the basis for identification of the etiology. In this study commonest etiology is acute CNS infection in all age groups of children. This is also supported by other[7,18,79] studies. It is in sharp contrast to adult hospital based studies, where cerebrovascular pathologies were predominant.

Both Hypoxic ischemic encephalopathy and epilepsy causing prolonged seizure activity are equally the second commonest cause of Impairment of consciousness and coma (non-traumatic) in our study. Epilepsy as the second commonest cause of non-traumatic coma is comparable with studies in Indian and other studies[36,67,74]

HIE is one of the important etiological category for impairment of consciousness and coma in children and it was supported by other studies[7,18,79].

The present study observed sex distribution and association of sex with etiology. CNS infections were common among both sexes. Results of sex distribution and association with etiology statistically showed no gender preference in distribution and specific etiological inclination towards any gender statistically. Other studies[18,24,36,79] also have similar view about relationship of sex with etiology.

In present study distribution of etiological causes are as below between 01 month and 3 years of age group had infection 35-31% , epilepsy, 14.58%, hypoxia 41.6%, between 4-5 years infection 35-41%, epilepsy 10%, hypoxia 9.375%, and between 6-12 years infection 50%, seizures 20%, metabolic 10% and others 15% Wong[24] et at and fabria[64] et at studies shows comparatively similar occurrence with our findings as

infection is the commonest and epilepsy is the second main cause in all age groups of children. Our study is comparable with Seshia[51] et al observation on age specific etiology.

In this present study patients admitted with presenting complaints both constutional and focal, non-focal neurological symptoms. Poor activity, refusal to feed, nausea and vomiting are the most common general symptoms presented in all age group and all categories of etiology Non-focal neurological symptoms like drowsiness, headache, neck pain, confusion were present in 6-12 years age group. Most of the CNS specific symptoms were presented in infective etiology. Other[24,64] studies shows similar presenting symptoms as our study observed.

In this study, duration of impairment of consciousness and coma in children was observed as 24% for $\bar{<}$ 1 days, 32% for $\bar{<}$ 3 days 35% for $\bar{<}$ 7 days 6% for $\bar{<}$ 14 days and 3% for >14 days Duration of coma was more prolonged mostly in acute CNS infection eg. Viral encephalitis, TB meningoencephalitis, than other causes. Duration of IC/C was shorter in most of the seizure disorder in children. Duration of coma was variable in hypoxic ischemic, metabolic, toxic and other etiologies. Other[24,36,79] studies have similar views with our study about relationship of duration of IC/C with etiology.

Patients presented with meningism 32% (with focal sings 12%) and without focal signs and meningism 68% Menigeal and focal sings were mostly present in the CNS infection Arun Bansal[7] et al and Wong et al[24] study is comparable with our findings as presence or absence of menigeal sings are important clinical clues.

The present study observed presence and absence of seizure due to specific etiology in non epileptic group of patients and its association with etiology. Seizures with specific etiology are due to arising neurological insult which cause reversible or irreversible irritable focal or multifocal epileptogenic region in the brain. Presence or absence of seizures with specific etiology with consideration of other clinical findings narrows down the diagnostic possibilities. Most of the seizures with specific etiology were present in diffuse or focal structural involvement in brain eg. viral encephalitis, Tb meningoencephalits, Tuberculoma, Neurocysticercosis. Seizure were present in hypoxic, metabolic, toxic case but lesser degree than CNS infection group. Arun Bansal[7] et al and other studies [12,48,64] emphasis the importance of seizures in IC/C patients as a reliable clinical predictor.

In present study CT brain imaging was done for 63 patients Selection of CT brain imaging was based on history and clinical examination. CT Brain positive patients were 23 and 40 were negative. CT Brain with positive findings were mostly related with acute CNS

infection tuberculoma and neuroysticercosis. CT Brain with normal result were more in non-infective etiologies. But CT negative findings did not rule out the infective etiology. Available studies did not focus on this important clinical variable.

Patients with IC/C and coma due to infection and seizures had good outcome compared with hypoxic-ischemic patients. Infective etiology patients had outcome as category I 68.42% II 9.09% III 2.63% IV 10.52% V 10.62% seizure patients had outcome as cat I 66.6 II 19.04 III 14.28% IV 0V0 Hypoxic ischemic patients had outcome as cat I 60.86 11 8.69 III 0V0 V 30.43% Metabolic, Toxic and other etiological group were very small in number and did not reach any meaningful statistical significance. In present study outcome by etiology is comparable with Arun bansal et al and other studies[7,64,79].

01 month - 3years age group patients had out come cat I 72.91% II 10.41% III 2.08% IV-0 V-14.58%, 4-5 years age group had outcome cat I 62.6% II 12.5% III-6.25% IV- 6.25% V.9.375% and 6-12year age group had I – 55% II -10% -III 5% IV -10% V-20% . All three age groups had variable outcome due to heterogenous group of multiple etiologies and different grades of severity and duration of IC/C. Overall assessment of association between age and outcome were not statistically significant.

Male patients had outcome as I -67.62% II-11.32% III-2.7% IV-1.88 V-15.09% Female patients had outcome as I- 65.95% II -10.6% III-

4.25% IV -6.38% V-12.76% This present study observed no significant gender preference in outcome. Other studies have similar analysis[24,36,67,79] and views about association of age and sex with outcome.

01 month -3 years age group patients had good outcome in infection and seizures and poor outcome in hypoxic-ischemic patients, 4-5 year age group patients had better outcome in infection and seizures compared with HIE , 6-12years age group patients had better outcome in infection and seizures and variable outcome with metabolic, toxic and other etiologies.

This study is comparable with other studies about relationship with age-specific etiology and outcome [7,64,79].

Patients with focal sings and meningism had poor outcome compared with patients who had no meningism and focal signs. Other[18,67,79] studies predict meningism and focal signs as unfavorable in IC/C patients.

This present study observed that patients with shorter duration of IC/C had better outcome compared with patients with longer duration of IC/C. Patients with long duration of impairment of consciousness had unfavorable outcome (Kirkham)$_{11}$.

Other [12,67,79] available studies assessed the duration of coma as an importation clinical factor to predict the prognosis.

This present study observed CICS/MGCS Score are the relatively reliable indicator for assessment of seventy of IC/C. Patients had $\bar{<}$ 8 GCS/MGCS Score had poor outcome compared with >8. Mortality rate progressively increased with decreased GCS score. Arun bansal et al and other[7,24,51] studies have the same conclusion about GCS Score.

This study observed outcome in patients who had multiple and prolonged seizures and who had no seizures in non-epileptic etiologies – CNS infection, hypoxic, metabolic, toxic and other causes. Secondary seizures were present more common in CNS infection than other groups. Symptomatic secondary seizures were due to heterogenous nature of multiple etiology of IC/C in children. Early identification and treatment of underlying etiology is the scientific choice and it will give better outcome along with controlling seizures also. Overall assessment of association of secondary seizures and outcome had significance. This study views have comparable with other studies[48,67,79] about seizures with specific etiology and outcome.

CT brain is one of the important neuroimaging diagnostic tool to find out the etiology. This study observed patients who had positive findings in CT brain did not show any statiscally significant difference in outcome compared with the patients who had normal findings in CT-

Brain. Other studies did not observe CT- brain results as a clinical variable for outcome impairment of consciousness and coma. This factor needs to be confirmed by further larger studies.

CONCLUSION

1. CNS infection is the commonest etiology and epilepsy is the second common etiology in all age groups and both sexes.
2. Low age groups are more susceptible for occurrence of impairment of consciousness and coma. Hypoxic – ischemic encephalopathy is the common etiology in 01 month to 3 year age group.
3. Gender had no role in etiological diagnosis and outcome.
4. Meningeal and focal signs are viable and valid factors to facilitate the diagnosis of etiology.
5. Severity of coma, duration of coma and meningeal and focal signs are the influencing factors upon the nature of outcome.
6. Secondary symptomatic seizures are more common in CNS infections than in other non epileptic etiologies. Presence of secondary symptomatic seizures in non-epileptic group had adverse impact on the outcome.
7. CT Brain with positive findings is an objective collateral evidence to diagnose etiology. Presence and absence of findings in CT brain is not significant in determining the outcome in this study. Further larger studies are needed.
8. Epilepsy had better outcome than other etiology.
9. Hypoxic ischemic, Metabolic, toxic and other etiological group had variable outcome due to heterogenous nature of multiple etiologies.

SUMMARY

In pediatric population, impairment of consciousness is an urgency and coma is an emergency. Proper history taking, focussed clinical examination and relevant laboratory investigations are essential to find out etiology. The present study shows that clinical variables such as age specificity, severity of coma, duration of coma, meningeal and focal signs and disease related seizures are important factors that can influence the outcome of non traumatic coma in paediatric population.

Evaluation and outcome of Impairment of consciousness and Coma in Pediatric Population

Data Entry form

1.	Serial No	:	
2.	Name	:	
3.	Age	:	
4.	Sex	:	
5.	H/o. Presenting illness	:	
	a) Non –Specific:		Fever, nausea , vomiting ,incessant cry, poor activity, refusal to feed
	b) CNS Complaints	:	Headache, neck pain, Confusion, irrelevant talk, seizures, focal weakness
6.	Conscious state	:	Irritable / drowsy / coma
7.	GCS/MGCS score	:	
8.	Duration of IC/C	:	
9.	Presence or Absence of	:	1) Meningeal signs 2) focal signs
10.	Seizures with specific etiology a) Present or Absent b) If present, type of seizures		GTCS / focal / subtle / NCS
11.	CT Brains + / - / Not taken		
12.	Etiological diagnosis	:	
13.	Outcome	:	CAT I, II, III, IV, V

PROFORMA

EVALUATION AND OUTCOME OF IMPAIRMENT OF CONSCIOUSNESS

AND COMA (NON-TRAUMATIC) IN PEDIATRIC POPULATION

Basic Information :

Name	Age	Sex	Height
Address		Income	Weight
			Head
circumference			
			Birth weight

Admitted with complaints of

History of Present illness: Onset : Acute, Sub acute, chronic, acute on chronic illness. Before IC/LOC, during IC/LOC After IC/LOC Headache, Fever, Fits, Confusion, Speech, Eye Closure, Excessive Sleepiness, Sleep Disturbance, Irritability. Restlessness, Refusal of Feeds, Vomiting, Inconsolable Cry, failure to cry even after Painful Stimuli, Neck Pain, Neck Tilt, Skin Rashes, Vesicles, Falls, Injury, Abdominal Pain, involuntary-movements, gait-disturbances, specific complaints while waking-(jerks, seizures)ASOM, CSOM, change of color of urine and motion. Toxin Exposure,History/Accidental Poisoning.	
Past History : Febrile and afebrile fits, meningoencephalitis, primary complex, recurrent respiratory and skin infection, Measles, Mumps, chicken pox, dog bite , recent vaccination. Chronic drug intake for any particular illness, rheumatic heart disease, congenital heart disease, recurrent syncope, joint swelling and pain, previous hospitalization.	

Family History : Headache, (Migraine) fits,contact Tb, Diabetes Mellitus, Hypertension, history suggestive of Neuro-developmental, neurodegenerative, neuro-inborn error of metabolic disease.	
Perinatal History : Birth weight term/premature, normal delivery/LSCS, Birth Asphyxia, cry immediately or delayed after birth, urine and meconium passed normally or not, Neonatal seizures, Neonatal hospitalization, flobby or Normal tone, color of the baby.	
Feeding History : Breast feeding or not, BF for how many months	
Developmental History : Motor, language, cognitive, social adaptive mile stones-Normal/delayed specific (or) global development. Bladder/Bowel Control, Toilet Behavior,	
Immunization History : Fully/partially/Not-according to the age.	
Treatment History :	

General Examination :

Vital Parameters	State of Consciousness	Congenital Anomalies
Temp	Febrile	
Pulse Rate	Hydration Status	Crania Facial
Blood Pressure	Anemic	
Anomalies		
Heart Rate	Jaundice	Spinal
Respiratory rate	Cyanosis	Neurocutaneous Markers
Extremities	Clubbing	Skin rash, Ulcer
Warm/Cold, Clammy	Odema	Hypo pigmented Patches

Urine Output Lympadenopathy External Injuries (Skull, Spine)

Oral Cavity : Color of Tongue, Ulcer Vesicles in Oral cavity.

No.	➢ 5 Years	< 5 Years
	Eye Opening :	
4	Spontaneous	
3	To Voice	
2	To Pain	
1	None	

Neurological Profile :

Glasgow Coma Scale

	Best motor Response :	
6	Obeys commands	Normal Spontaneous movement.
5	Localises to Pain	
4	Normal Withdrawal	
3	Abnormal flexion	
2	Abnormal extension	Alert, Normal words or babbling or cooing.
1	None	Less than usual, irritable cry.
		Cries to pain.
	Best Verbal Response :	Mons to pain.
5	Oriented	No Response.
4	Confused	
3	Inappropriate words	
2	Incomprehensible Sounds	
1	None	

<u>Grades of Coma :</u> Stage

Signs of meningeal Irritation :

1. Neck Rigidity 2. Kernig's Sign 3. Brudzinski-neck sign

Brainstem Reflexes :

1. **Pupils :** Left Right

   Size

   Position

   Reactivity to Light

2. **Spontaneous Eye Movements :**

   Orienting

   Roving Conjugate

   Roving Dysconjugate

   Miscellaneous Abnormal Movements

   None

3. **Oculocephalic Response :**

   Full

   Minimal

   None

Oculovestibular Response :

Normal

Tonic Conjugate

Minimal or Dysconjugate

None

Corneal Reflex

Gag reflex

Respiratory Pattern

Focal neurological deficit

Other Signs :

Presence of yawning, swallowing, licking movements of the lips.

Hiccup present or absent.

Bulging Anterior Fontanelle.

Decorticate rigidity.

Decerebrate rigidity.

Skeletal muscular tone.

Deep Tendon Reflexes.

Plantar Reflex.

Evidence of seizure Activity : (Convulsive /Non Convulsive)

(Focal eye deviation, Nystagmus, eyelid and focal twitching, unilateral clonus, Head deviation, Rhythmic Myoclonus, Cycling movement, lip smacking)

Look for Evidence of Herniation :

Changing pattern of pupils, Eye movements, respiratory pattern III, VI Cranial Nerve palsy (Unilateral, bilateral) and others.

Presence of Cushing Triad :

Bradycardia, Hypertension, Respiratory Pattern.

Evidence of Metabolic /fluid/Electrolyte / Acid-base imbalance

Hydration Status.

Non-Neurogenic Hyper or Hypoventilation.

Astrexis.

Myoclonus.

Tremor.

Urine out put.

Evidence of Acute /chronic and other systemic illness.

Provisional Diagnosis

Final Diagnosis

Investigations :

Urine-Albumin, Sugar, Deposits

Complete blood count

ESR-BT-CT

Peripheral smear for blood picture/malaria parasite.

Blood sugar.

Serum Creastine.

Serum Electrolytes-Sodium Potassium.

Liver Function Test.

Chest X-ray PA view.

ECG

CSF Analysis : Cells	Biochemical Analysis	Culture and Sensitivity
	Sugar	
	Protein	
	Chloride	Others

CT Brain

MRI Brain

EEG

Neurosonogram

Others (USG-Abdomen)

Prognosis and Outcome :

Examination	**After**	**Day**	**D**	**D**	**D**	**D**
1. Status of Consciousness (GCS)	6 Hours	1	3	7	15	30
2. Brainstem reflexes						
(a) Pupil Size						
(b) Reactivity to light						
3. Respiratory Pattern						
4. Focal neurological deficit						
5. Seizure Activity						
6. Evidence of Herniation						
7. Presence of Yawning, Swallowing ,licking Movements of the lips.						

Grades of Outcome of Coma :

			After 6 Hrs	Day 1	D 3	D 7	D 15	D 30
1.	Good Recovery	Patients who regain the ability to conduct a normal life or, it a preexisting disability exists, to resume the previous level of activity.						
2.	Moderate disability	Patients who achieve independence in daily living but retain either physical or mental limitations that preclude resuming their previous level of function.						
3.	Servere disability	Patients who regain at least some cognitive function but depend on others for daily support.						
4.	Vegetative state	Patients who awaken but give no sign of cognitive awareness.						
5.	No recovery	Patients who remain in coma until death.						

KEY TO MASTER CHART

Age	:	M-Months
		Y-Years
Sex	:	M-Male
		F-Female
Etio	:	Etiology
I	:	Infection
E	:	Epilepsy
H	:	Hypoxic Ischemic
M	:	Metabolic
T	:	Toxic
O	:	Others
Dur	:	Duration
CS	:	Conscious State
I	:	Irritable
D	:	Drowsyness
C	:	Coma
GCS	:	Glasgow Coma Scale
T	:	Temperature
PR	:	Pulse Rate
BP	:	Blood Pressure
RR	:	Respiration Rate

CO	:	Conjuctival Reflex
CR	:	Corneal Reflex
DEM	:	Dolls eye movements
RP	:	Respiratory Pattern
MP	:	Motor Pattern
Fs/M	:	Focal and Meningeal Signs
Sec Seizures	:	Secondary Seizures
LP	:	Lumbar Puncture
MR	:	Magnetic Reasonace imaging
EEG	:	Electro encephalogram
CBC	:	Complete blood count
LFT	:	Liver function test
RFT	:	Renal function test
G	:	General
F	:	Focal
S	:	Subtle
CT	:	Computed Tomography
Dia	:	Diagnosis

Introduction

Review of Literature

Aims of the Study

Materials and Methods

Observation, Analysis & Results

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Appendix

Proforma

Master Data Chart

MASTER DATA CHART

Sl.No	Age	Sex	Durat (days)	CS	CGS	T	PR	BP	RR	CO	Cr	DEM	RP	MP	FS/M	Sec Seizure	LP	CT	MRI	EEG	CBC	LF	RFT	Diag	Etiolo	Out come
1	04M	M	7	I	12	AB	AB	AB	AB	N	N	N	N	N	N	G	P	N	-	-	-	-	-	BM	I	1
2	9Y	M	3	D	13	N	N	N	N	AB	AB	N	N	AB	N	SD	N	P	P	AB	N	N	N	CH	E	3
3	6M	M	7	I	11	AB	AB	AB	AB	N	N	N	N	N	N	G	P	N	-	-	-	-	-	BM	I	1
4	10Y	F	30	D	10	AB	AB	N	AB	AB	AB	AB	N	AB	M	G	P	P	-	-	N	N	N	TBM	I	4
5	5Y	M	30	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	M	G	P	N	-	N	N	N	N	TBM	I	5
6	2Y	M	14	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	M	G	P	P	-	-	-	-	-	TBM	I	3
7	5Y	M	3	D	13	AB	N	N	N	N	N	N	N	N	N	SD	-	N	-	-	-	-	-	SE	E	1
8	11M	M	3	I	12	AB	AB	AB	AB	N	N	N	N	N	N	G	P	P	-	-	-	-	-	BM	I	1
9	8Y	M	14	D	10	AB	AB	N	N	AB	AB	AB	N	AB	FS	G	AB	N	P	-	N	N	N	VE	I	4
10	9Y	M	7	I	12	N	N	N	N	N	N	N	N	N	M	F	N	P	-	-	-	-	-	NCC	I	1
11	3Y	F	1	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	P	-	-	-	SE	E	1
12	11Y	M	1	D	13	N	N	N	N	AB	AB	N	N	AB	N	SD	N	N	P	AB	N	N	N	SE	E	1
13	07M	F	7	D	13	AB	AB	AB	AB	N	N	N	N	N	N	S	N	TN	-	-	-	-	-	BN	H	1
14	4Y	M	3	D	13	N	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	N	N	UP	T	1
15	9Y	F	7	D	12	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	AB	HE	M	5
16	15M	M	3	I	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	P	-	-	-	-	-	BM	I	1
17	11Y	M	14	I	11	AB	N	N	N	AB	AB	N	N	AB	FS	G	AB	P	-	-	N	N	N	TBM	I	2
18	3Y	F	1	D	12	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	2
19	4Y	F	14	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	M	G	P	N	P	-	-	-	-	HSE	I	4
20	11M	M	3	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	S	-	TN	-	-	-	-	-	CHD	H	5
21	18M	M	3	I	12	AB	AB	AB	AB	N	N	AB	N	N	N	G	P	P	-	-	-	-	-	BM	I	1
22	4Y	M	3	D	13	N	N	N	N	N	N	N	N	N	N	N	NT	N	-	-	-	AB	N	HE	M	1
23	9M	F	3	I	12	AB	AB	AB	AB	N	N	N	N	N	N	G	P	P	-	-	-	-	-	BM	I	1
24	5Y	F	1	D	13	AB	N	N	N	N	N	N	N	N	N	SD	-	N	-	-	-	-	-	SE	E	1
25	4M	F	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	N	TN	-					BN	H	1
26	6M	M	1	D	13	AB	AB	AB	AB	N	N	N	N	N	M	N	-	TN	-	-	-	-	-	BN	H	1
27	6Y	M	3	D	8	AB	AB	AB	AB	AB	AB	N	AB	AB	N	N	-	TN	-	AB	-	-	N	LUK	O	5
28	6M	F	1	D	13	AB	AB	AB	AB	N	N	AB	N	AB	N	S	-	N	-	-	-	-	-	BN	H	1
29	5Y	F	7	C	8	AB	AB	AB	AB	N	AB	N	AB	AB	M	G	P	N	P	-	N	N	N	VE	I	4
30	3Y	F	1	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
31	12Y	M	30	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	FS	F	P	P	-	-	N	N	N	TBM	I	5
32	4Y	F	7	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	M	G	N	N	-	-	AB	AB	AB	SCF	H	5
33	16M	F	3	I	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BM	I	1
34	4Y	M	3	D	13	AB	AB	AB	AB	AB	AB	N	AB	N	N	N	-	TN	-	-	-	-	-	SS	T	1
35	07M	M	1	D	13	N	N	N	N	N	N	N	N	N	M	N	-	TN	-	-	-	-	-	BN	H	1
36	5Y	F	7	I	13	AB	AB	AB	AB	N	N	N	N	N	FS	N	P	P	-	-	-	-	-	TBM	I	2
37	18M	F	3	I	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BM	I	1
38	4Y	F	1	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
39	3Y	F	3	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	AB	AB	-	HE	M	1
40	13M	M	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	G	-	TN	-	-	-	-	-	BN	H	1
41	11Y	M	7	I	13	AB	N	N	N	N	N	N	N	N	M	G	-	N	P	N	N	N	N	CVT	O	2
42	1Y	F	3	D	13	AB	AB	AB	AB						M	N	-	TN	-	-	-	-	-	CHD	H	2
43	7Y	M	7	I	12	N	N	N	N	N	N	N	N	N	N	F	N	P	N	N	N	N	N	NCC	I	1
44	06M	M	3	D	12	AB	AB	AB	AB	AB	AB	AB	A	AB	N	S	-	TN	-	-	-	-	-	BN	H	5
45	3Y	M	14	C	8	AB	AB	AB	AB	AB	AB	AB	A	A	N	G	P	N	P	-	-	-	-	VE	I	5
46	4Y	M	7	I	12	N	N	N	N	N	N	N	N	N	M	F	-	P	-	-	N	N	N	TBM	I	1
47	15M	M	3	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	SS	T	1
48	13M	F	3	I	12	AB	AB	AB	AB	N	N	N	N	N	N	G	P	N	-	-	-	-	-	BM	I	1
49	4Y	F	1	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
50	15M	M	3	C	8	AB	AB	AB	AB	AB	AB	AB	A	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
51	03M	M	7	D	13	AB	AB	AB	AB	N	N	N	N	N	S	-	TN	-	-	-	-		BN	H	1
52	4Y	M	7	I	12	N	N	N	N	N	N	N	N	N	M	F	-	P	P	-	N	N	N	NCC	I	1
53	15M	F	7	D	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BN	I	1
54	8M	M	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	SA	H	1
55	4Y	M	3	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	AB	N	SB	T	1
56	2Y	F	3	D	13	AB	AB	AB	B	N	N	N	N	N	N	N	-	TN	-	-	-	-	N	CHD	H	2
57	4Y	F	7	D	13	N	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	N	TN	-	P	-	-	-	SE	E	2
58	13Y	F	7	D	12	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	N	NB	TN	-	-	-	-	-	SA	H	5
59	5Y	M	3	I	12	N	N	N	N	N	N	N	N	N	M	F	-	P	P	-	-	N	N	NCC	I	1
60	09M	M	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	SA	H	1

Sl.No	Age	Sex	Durat (days)	CS	CGS	T	PR	BP	RR	CO	Cr	DEM	RP	MP	FS/M	Sec Seizure	LP	CT	MRI	EEG	CBC	LF	RFT	Diag	Etiolo	Out come
61	6Y	F	7	I	13	AB	N	N	N	N	N	N	N	N	FS	F	N	P	N	-	N	N	N	TUB	I	1
62	5Y	F	7	D	10	AB	AB	AB	AB	AB	AB	AB	AB	AB	M	G	N	N	-	-	AB	AB	AB	SEF	H	5
63	6Y	M	1	D	13	N	N	N	N	AB	AB	N	AB	AB	N	G	-	N	-	-	-	-	-	UP	O	1
64	5Y	M	7	I	12	AB	AB	AB	AB	N	N	N	N	N	M	F	-	P	-	-	AB	N	-	CAB	I	1
65	11M	M	7	D	13	AB	AB	AB	AB	N	N	N	AC	N	N	G	N	TN	-	-	-	-	-	BN	H	1
66	10Y	M	1	D	13	N	N	N	N	N	N	N	N	AB	N	SD	N	TN	N	N	N	N	N	SE	E	1
67	8M	F	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	BN	H	1
68	5Y	M	3	D	13	N	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	AB	-	AB	AGN	M	1
69	3Y	F	7	D	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BM	I	1
70	5Y	M	7	D	12	N	AB	AB	AB	AB	AB	AB	A	AB	N	SD	-	TN	-	P	-	-	-	SE	E	2
71	2Y	F	3	D	13	AB	AB	AB	N	N	N	N	N	N	N	N	-	TN	-	-	-	-	AB	AGN	M	1
72	12M	M	3	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
73	5Y	F	7	I	12	AB	AB	AB	AB	N	N	N	N	N	FS	F	-	P	-	-	AB	-	-	CAB	I	1
74	3Y	F	3	C	8	AB	AB	AB	AB	AB	AB	AB	A	A	N	G	P	N	P	-	-	-	-	VE	I	3
75	9Y	F	7	D	10	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	N	AB	N	HE	M	5
76	14M	M	7	D	12	AB	AB	AB	AB	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BN	I	1
77	7Y	M	7	I	13	AB	N	N	N	N	N	N	N	N	N	F	N	P	N	N	-	N	N	CAB	I	1
78	04M	M	7	I	12	AB	AB	N	N	N	N	N	N	N	M	G	P	N	-	-	-	-	-	BM	I	1
79	5Y	F	3	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	SS	T	1
80	5M	F	7	D	12	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	S	-	TN	-	-	-	-	-	BN	H	5
81	5Y	F	7	I	12	AB	N	N	N	N	N	N	N	N	N	N	-	N	-	-	-	-	-	BM	I	1
82	9M	M	7	I	12	AB	AB	AB	AB	A	A	N	N	N	N	G	P	N	-	-	-	-	-	BM	I	1
83	12Y	F	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	G	N	TN	-	-	-	N	N	UP	T	1
84	11Y	F	7	I	13	AB	N	N	N	N	N	N	N	N	FS	F	N	P	N	N	N	N	N	TUB	I	1
85	9Y	F	1	D	13	N	N	N	N	N	N	N	N	N	N	SD	N	N	N	N	N	N	N	SE	E	1
86	5Y	F	7	C	8	N	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	N	N	N	N	P	N	N	SE	E	1
87	5M	F	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	-	TN	-	-	-	-	-	BN	H	1
88	5Y	F	3	D	13	N	N	N	N	N	N	N	N	N	N	N	N	TN	P	P	P	N	N	ME	M	2
89	10M	M	1	D	13	AB	AB	AB	AB	N	N	N	N	N	N	N	NT	TN	-	-	-	-	-	SA	H	1
90	4Y	F	7	D	13	N	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	-	-	-	-	SE	E	2
91	6Y	M	7	D	13	AB	N	N	N	N	N	N	N	N	FS	N	AV	P	N	N	N	N	N	TBM	I	1
92	18M	M	3	C	8	AB	AB	AB	AB	AB	AB	AB	AV	AB	N	SD	-	N	-	-	-	-	-	SE	E	1
93	4Y	F	1	D	13	AB	AB	AB	AB	N	N	N	AV	N	N	N	-	TN	-	-	N	N	N	SA	H	1
94	4Y	F	7	C	8	N	AB	AB	AB	AB	AB	AB	AV	AB	N	SD	-	N	-	P	N	N	N	SE	E	3
95	2Y	M	14	C	8	AB	AB	AB	N	AB	AB	AB	AB	AB	N	G	P	P	-	-	N	N	N	TBM	I	1
96	4Y	M	3	D	13	N	AB	AB	N	N	N	N	N	N	N	N	-	TN	P	-	AB	-	Ab	AGN	M	5
97	2Y	M	3	D	13	N	N	N	N	N	N	N	N	AB	FS	G	-	P	-	-	-	-	-	IH	O	2
98	4Y	M	1	D	13	AB	N	N	AB	N	N	N	N	N	N	SD	-	N	-	P	-	-	-	SE	E	1
99	2Y	F	3	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	SD	-	N	-	P	-	-	-	SE	E	1
100	09M	M	1	C	8	AB	AB	AB	AB	AB	AB	AB	AB	AB	N	G	-	TN	-	-	AB	N	N	CHD	H	5

CT BRAIN COMPLICATION OF TBM NON COMMUNICATING HYDROCEPHALUS

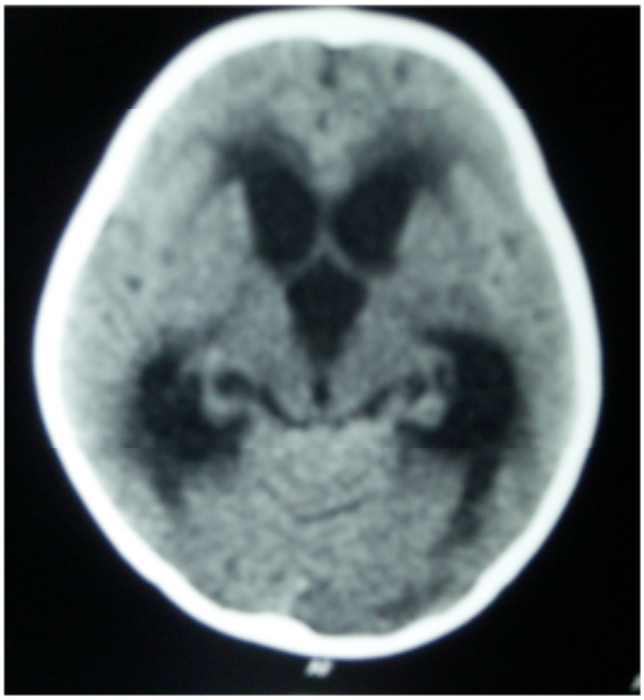

FIG:1

BRAIN ABSCESS WITH LARGE PERILESIONAL OEDEMA AND MIDLINE SHIFT TO THE LEFT

FIG:2

CECT BRAIN SUBBDURAL EMPYEMA IN THE RIGHT FRONTOPARIETAL AND LEFT FRONTAL REGIONS (CURVILINEAR RING ENHANCEMENT SEEN)

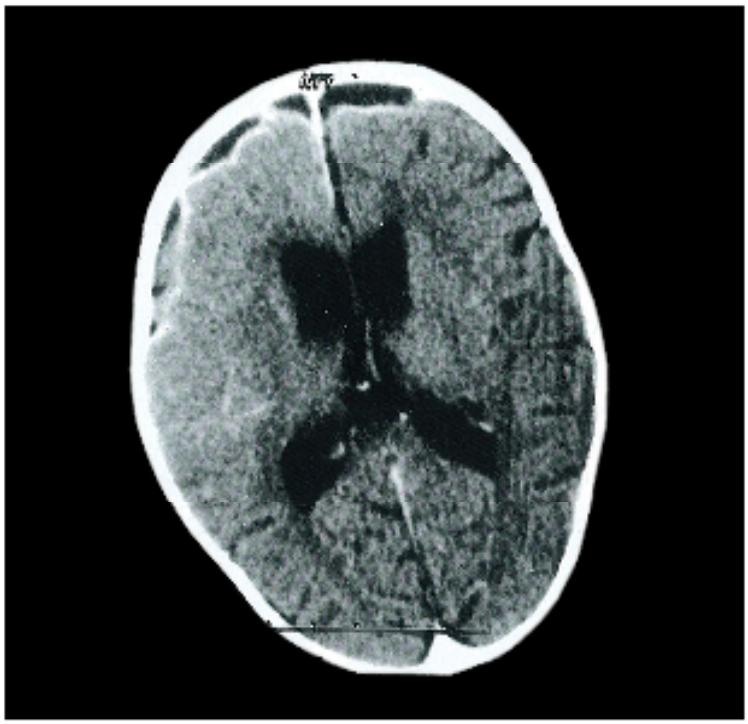

FIG:3

A CASE OF TB MENINGOENCENPHALITIS

A CASE OF BACTERIAL MENINGITIS

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